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BACILLARY DYSENTERY IN AN AUSTRALIAN HOSPITAL IN THE MIDDLE EAST.

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THE following account of bacillary dysentery occurring in Australian, New Zealand and British troops admitted to an Australian General Hospital is based on our observations in the dysentery wards during the spring and summer of 1941. No novel views are advanced; but as bacillary dysentery is uncommon in Australia, it is felt that a description of the clinical features and management of these cases may be useful. Many of the 600 patients admitted to the wards had been treated elsewhere during the acute stage and reached us when convalescent. A few cases of amœbic dysentery were seen. The scope of this paper will be restricted to some 300 cases of diarrhœa arising among troops situated in the vicinity of the hospital and seen within the first few days of the illness.

Description of Wards and Precautions against Cross-Infection.

The sites of the wards were chosen and their construction was modified with the objects of minimizing cross-infection within the wards, of preventing spread of the disease to other wards and departments of the hospital, and of facilitating prompt examination of the stools in the laboratory. The two dysentery wards were sited as near as possible to the laboratory. They were wire-screened against flies. The annexes, situated in the centre of each ward, were modified, so that an enclosed wire-screened room was provided in the rear. In this room, a large wire-screened cupboard was built to hold the bed pans containing stools awaiting inspection by the medical officer. The used pans were thus doubly shielded from flies. An incinerator was built within the room so that fæces could be destroyed without leaving the dysentery ward. The fæces were mixed with sawdust and burnt in the incinerator; this procedure gave rise to no objectionable smell. Two coppers were kept boiling beside the incinerator in which bed pans were sterilized before being returned to wire-screened cupboards in the wards. The latrines for ambulatory patients had their back opening into the incinerator room, so that the pans could be emptied directly into the incinerator. All linen was boiled before it was sent to the hospital laundry. Printed rules and instructions regarding personal hygiene and the risks of flies were given to all patients on their admission to the wards and reinforced by talks during the daily round. Naturally, in spite of these precautions, cases of bacillary dysentery did occur amongst the staff and patients of other wards; but it is unlikely that the source of infection was the dysentery wards. Only one probable case of cross-infection was detected within the dysentery wards: a patient convalescent from amœbic dysentery contracted a bacillary infection, Flexner type V. Another patient with a type V infection, which is uncommon, was in the ward at the same time.

Selection of Stools for Bacteriological Examination.

Stools were sent for laboratory examination on the first three days after admission to the wards, and thereafter as ordered by the physician according to the condition of the patient and the characters of the stool. The dysentery wards were almost immediately adjacent to the laboratory, so that delay in transit of the specimen was minimal. The whole stool in the pan was not sent. The orderlies, permanently attached to the wards, were trained to select a suitable sample and to bring it immediately to the laboratory. A long-handled copper spoon was used to obtain the specimen from the pan. It was sterilized after

the taking of each sample by immersion in boiling water. To check the suitability of the sample and the promptitude of dispatch, a form was designed to be forwarded with each specimen. On this form the orderly described, by crossing out inapplicable terms, the gross appearance of the fæces and the exact time at which it was passed. In the laboratory, the macroscopic characters of the specimen were entered on the same form, and the time of receipt was noted. The orderlies quickly became conversant with their duties. There were seldom other than minor discrepancies between the descriptions of the whole stool and of its sample, and only exceptionally was there an interval of over ten minutes between the passage of the stool and the receipt of the sample at the laboratory.

Laboratory Examination of Stools.

Part of the specimen, if suitable, was incubated as soon as it was received, in order to obtain a culture; the remainder was then examined microscopically. A standardized nomenclature was used to describe the gross and microscopic appearances. For the macroscopic features suitable combinations of the terms "solid", "soft", "liquid", "blood and mucus", "mucus only", "no mucus" sufficed. The microscopic findings were described in the standard army terms "typical bacillary exudate", "indefinite exudate", or "no exudate"—according to the presence or absence of red blood corpuscles, polymorphonuclear cells, macrophages and columnar epithelial cells. A typical bacillary exudate usually contains all four types of cell, though red cells may be absent and epithelial cells are not invariably present. The mucus in the acute case viewed by naked eye after having been washed in saline solution is typically a small, flat, thin, semi-transparent piece of tissue, which microscopically appears to be a sheet of superficial bowel epithelium in which most of the epithelial cells have been replaced by polymorphonuclear cells. Or the exudate may consist of mucoid secretion in which the numerous cells are embedded but separated by mucus. This latter type of exudate grades into the "indefinite" class if cells are rare and the intercellular mucus is abundant. In liquid stools containing no mucus there may be varying numbers of polymorphonuclear cells, perhaps a few macrophages, rarely red cells or epithelial cells. Such an exudate will usually be classed as "indefinite". The tough mucus, often blood-tinged, seen on the surface of solid or scybalous stools, consisting of closely packed squamous epithelial cells perhaps with a few polymorphonuclear cells and varying numbers of red cells, is not to be confused with a dysenteric exudate.

The Growing of Culture from Stools.

A small piece of suitable mucus is taken out with a hooked platinum wire, washed in two amounts of five cubic centimetres of normal saline solution until free of fæcal material and inoculated on a single three-inch MacConkey plate. In our limited experience MacConkey's medium made according to the original formula (Medical Research Council of the Privy Council, Special Report Series 51) is as satisfactory as other media and is easy to prepare. Except in cases of special interest (which included the first specimen from a newly admitted patient), culture was attempted only from stools containing mucus. We found, as many others of far wider experience have found before us, that dysentery bacilli were rarely isolated from fæces, even liquid fæces, but were constantly removed from the typical mucus present in a fresh stool. Dysentery bacilli were isolated from the various types of diarrhœic stools in the following proportions:

Stools with typical bacillary exudate, red cells present	90% (82 cases)
Stools with typical bacillary exudate, red cells absent	70% (43 cases)
Indefinite exudate, liquid stool, with or without mucus	23% (53 cases)
No exudate, liquid stool	14% (56 cases)

Even among soft formed stools, if mucus embedded in the fæces is recovered by emulsification of the stool in saline solution, a high percentage of successful isolations is the rule. This type of stool is usually encountered late in convalescence.

Appreciation of the fact that the presence of mucus is the best indication of whether attempted culture will be successful, and therefore of persistent infection, enables the physician to assess the patient's progress and to select suitable stools for further investigation. It is usually stated that if dysentery bacilli are to be isolated, the stool must be examined early in the disease. In so far as the acute stage of dysentery may be transient, this advice is sound. But in practice it has been found that successful culture is just as likely in the late as in the early stages, providing the patient continues to pass a stool which contains mucus and shows a typical exudate. Such stools may be passed intermittently, normal ones intervening. In this series the stools of 42 patients with exudates examined within forty-eight hours of the onset of the disease gave 31 positive cultures and 11 failures. The stools of 32 patients with exudates examined after forty-eight hours of the onset of the disease, but within fourteen days, gave 30 successful cultures and only two failures. The probable explanation of this somewhat paradoxical result is that the group examined within forty-eight hours of the onset included a greater percentage of patients with milder infections. The result of culture was usually available in the ward within eighteen to twenty-four hours of the patient's admission to the wards. Suspicious colonies were picked from the plates after incubation overnight and subcultured on agar slopes, and after a further five or six hours' incubation there was sufficient growth for slide agglutination.

Varieties of Dysentery Encountered.

The distribution of the various varieties of dysentery was as follows: *Bacillus dysenteriæ* Flexner 148, *Bacillus dysenteriæ* Shiga 13, *Bacillus dysenteriæ* Sonne 12, *Bacillus dysenteriæ* Schmitz 15.

Forty-eight strains were not agglutinated by the stock serum, of which 18 varieties fermented mannite and 30 did not. Of the mannite fermenters, some probably belonged to types not included in the stock pooled Flexner serum, particularly *Bacillus dysenteriæ* Boyd II. These strains were chiefly isolated early in the series, when serum for this type (which was later shown to occur in this area) was not available. The status of the inagglutinable, non-mannite fermenting strains is doubtful and is the subject of investigation elsewhere. It is believed that only a very small proportion of them are to be regarded as true dysentery bacilli.

Colonel J. S. K. Boyd kindly supplied monovalent serum by which the various types comprising the Flexner group could be identified. A number of strains had been discarded when these specimens of serum were obtained, but 101 strains were typed as follows: *Bacillus dysenteriæ* Flexner I, 10; *Bacillus dysenteriæ* Flexner II, 55; *Bacillus dysenteriæ* Flexner III, 4; *Bacillus dysenteriæ* Flexner IV, 8; *Bacillus dysenteriæ* Flexner V, 5; *Bacillus dysenteriæ* Flexner VI, 8; *Bacillus dysenteriæ* Boyd I, 5; *Bacillus dysenteriæ* Boyd II, 3; *Bacillus dysenteriæ* Boyd III, 1; *Bacillus dysenteriæ* P274, 2. All the types established by Boyd were therefore encountered. We had originally hoped to collect sufficient cases of each type to establish whether or not characteristic clinical features are associated with particular types. It is obvious from the above-mentioned distribution of types that far too few cases of the individual types were seen to attempt clinical differentiation. The strains are distinct epidemic entities, and cases arising within a single ward of the hospital were always due to a single type.

Clinical Features of the Disease.

The onset of the attack is almost invariably signalled by colicky pain, shortly followed by urgent diarrhœa. These symptoms may be accompanied, preceded or followed by vomiting, and are usually associated with general malaise, headache, pain and aching in the muscles, and pyrexia. The relative incidence of the various symptoms in 62 proved cases of bacillary dysentery representing all types of infection encountered has been determined. In these cases a detailed history was taken and a full examination was made according to a scheme devised to

elicit exact information on controversial points. When the presence or absence of a particular symptom is not mentioned in the notes, that case has been excluded in the assessment of the incidence of the symptom.

Nausea and Vomiting.

Half the patients (29 of 60) complained of nausea preceding or accompanying the onset of diarrhœa, and one-third (19 of 59) vomited. Vomiting was seldom severe and never persistent. Twelve of these patients vomited only once, three vomited twice, and the remaining four vomited several times. In only four cases did vomiting precede the onset of the colic, in eleven it accompanied the onset, and in four it was somewhat delayed.

Headache.

About two-thirds of the patients complained of headache (36 of 59). In only four cases did the headache precede the onset of the diarrhœa by twelve hours or more, but in twelve the headache preceded the onset by a few hours.

General Malaise: Aching, Pain and Shivering.

About half the patients (27 of 53) complained of aching and pain in the limbs, joints and muscles. These symptoms preceded the onset of colic in eleven cases, were associated with the onset in ten, and developed later in six.

Pyrexia.

The great majority of patients admitted to hospital with acute dysentery were pyrexial; but obviously information is unobtainable as to whether this condition preceded the onset of the dysenteric attack.

Prodromal Symptoms.

If the above-mentioned symptoms are taken in combination, roughly one-third of the patients (17 of 53) had some prodromal symptoms, and in nine cases these preceded the onset of dysentery by twelve hours or more. Two patients were admitted to hospital with a diagnosis of "P.U.O." the day before the diagnosis declared itself.

There was no apparent association between the presence of definite prodromal symptoms and the severity of the attack of dysentery as judged by the intensity and duration of the diarrhœa. In fact, if anything, such symptoms were commoner in the milder cases. Possibly the attention of those patients with really acute diarrhœa and colic was so occupied with the more urgent symptoms that the milder systemic reaction was disregarded.

Abdominal Pain and Tenesmus.

Pain in the abdomen is almost invariably present, and is usually described as a constant dull ache with intermittent attacks of colic. Only three patients of 64 had no colic; two of the three complained of tenesmus, and in the other case the presence or absence of tenesmus was not recorded in the notes. Tenesmus on the other hand was rarely noted. It was present in eight of 35 cases. In the remaining 29 cases there is no record of its presence or absence.

Abdominal Tenderness.

Abdominal tenderness was present on admission to hospital in the great majority of cases (52 of 61). It was commonest in the lower part of the abdomen in the right and left iliac fossæ, next commonest in the upper part of the abdomen, and infrequent in the mid-abdomen. Tenderness was usually somewhat diffuse, but seldom generalized. The recorded frequencies of the various sites in 61 patients were as follows:

Upper part of the abdomen, 42:	
Right hypochondrium	16
Epigastrium	10
Left hypochondrium	16
Mid-abdomen, 21:	
Right lumbar region	8
Umbilical region	5
Left lumbar region	8
Lower part of the abdomen, 88:	
Right iliac fossa	33
Hypogastric area	22
Left iliac fossa	33

In most cases tenderness rapidly disappeared; in about half it persisted for a few days, usually settling down in one or both iliac fossæ. Persistent tenderness was twice as frequent in the left iliac fossa as in the right iliac fossa (14:7).

Diarrhœa.

The diarrhœa was usually explosive in onset, and a history of a few loose stools for two or three days succeeded by severe diarrhœa, though it did occur, was uncommon. More than half the patients had the greatest number of motions on the day the diarrhœa started. The figures for 52 patients are as follows:

Day of most numerous stools:

First day	30 patients
Second day	12 patients
Third day	6 patients
Fourth day	2 patients
Fifth day	2 patients
Ninth day	1 patient

There was a definite correlation between the severity of the attack at onset and the duration of the disease. The relevant data are shown in a scatter diagram (Figure I). Reference to this figure will show that the patients fell roughly into two classes: those with a maximum number of stools on any day not exceeding 15, whose diarrhœa lasted from three to six days, and those with the maximum number of stools exceeding 15, whose diarrhœa persisted from six to sixteen days. For the purposes

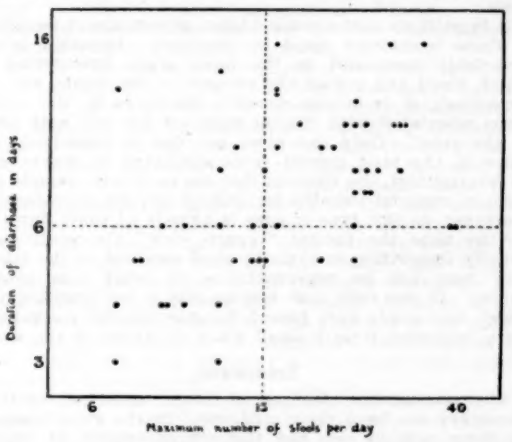

FIGURE I.

of this calculation diarrhœa was considered to have ceased when the number of stools fell permanently to three or less per day. Recrudescence of diarrhœa was rare when during treatment the number of stools fell to three. Persistence of diarrhœa for more than sixteen days was uncommon. It occurred in only four cases of this series of 62; two of the patients had an initial mild diarrhœa (maximum stools less than 15 per day from the onset), and two had severe diarrhœa at the onset, which subsided gradually, but persisted for a long while. The incidence of these persistent cases can be judged better from the fact that in 178 cases in which typed dysentery bacilli were isolated, there were six persistent cases in which there was strong evidence that the continued symptoms had been due to the organism isolated. These cases included two Shiga, two Schmitz and two Flexner IV infections. This incidence of 3·5% agrees with the experience of Fletcher and Mackinnon in the last war, who found that 2·8% of their patients (26 of 935) became persistent carriers.

Cases of Presumed Bacillary Dysentery in which Dysentery Bacilli were not Isolated.

Consideration of the foregoing description of the signs and symptoms in proved cases of bacillary dysentery

during this period shows that the typical case is one of acute diarrhœa, accompanied from the onset by colicky pain and often by vomiting which is never persistent, and associated with or preceded by mild general symptoms, pyrexia, often rigors, headache, aching of the limbs. The attack is usually explosive in nature, with the maximum number of stools on the first day; the patient's condition improves rapidly with rest and suitable diet.

Most of the cases of acute diarrhœa admitted to the wards conformed to this clinical picture. They were divisible into three classes, according to whether a typical bacillary cellular exudate, an indefinite exudate or no exudate was found in the stool. The first group, in which typical exudates were present, were undoubtedly cases of bacillary dysentery. The question is whether cases in which there was no definite exudate are to be placed in the same category.

For the following reasons we believe, in conformity with previous observers, that the vast majority of cases of acute diarrhœa occurring among adults in the Middle East are dysenteric infections. The clinical features resemble those of the proved cases, but the attack is usually milder and shorter. No organisms other than dysentery bacilli have been isolated from the stools in such cases. It was thought at one time that some or many of these cases of diarrhœa might be salmonella infections ("food poisoning"). Salmonella organisms have never been isolated in such cases in this hospital, whereas dysentery bacilli have been recovered in about 20% of them.

It is unlikely that a much higher percentage of isolations of these organisms could be expected. Isolation is not invariably successful in the most acute dysenteries, in which blood and mucus are present in the stools, and the percentage of isolations sensibly decreases in the milder cases associated with typical exudates but not with blood in the stool. Only one stool per day is examined, and since in the mild proved cases discharge of mucus may be intermittent, the chances that the particular sample will contain material suitable for culture are not considerable; moreover, as this type of case is usually of short duration, by the time the patient "reports sick" his condition is usually improving, so that the stool received at the laboratory may not be representative of what was present earlier. In one mild case lasting only a few hours, during which four stools were passed, Flexner dysentery organisms were recovered from a small piece of mucus in the stool.

Treatment.

The outstanding feature of these cases of bacillary dysentery has been their mildness. In the great majority of cases rest in bed and the administration of copious fluids and suitable diet lead to rapid recovery. Sodium sulphate was given in all acute cases, 120 grains on admission to hospital followed by 60 grains for three doses at intervals of two hours, then 60 grains every four hours; the daily dose is then gradually reduced by lengthening of the interval between administrations. At first sodium sulphate was given for seven to ten days after the patient's admission to hospital; later the course of treatment was considerably shortened (it seldom exceeded five or six days), as it was thought that in some cases the sodium sulphate caused the diarrhœa to persist. Our present practice is to give sodium sulphate every four hours for three or four days, according to the severity of the attack, and then to reduce the administration to one early morning dose.

Two types of diet were used. One of us (R.R.A.) restricted the diet to clear fluids and jelly until the stools were reduced to six to ten per day, at which stage toast and cereals were allowed. When the patient was having three or four daily motions, protein was added as well as fats in small quantities. A week after admission to the wards most patients were receiving the ordinary hospital light diet. The other diet (F.R.H.) differed only in so far as milk drinks were the first addition to the diet of clear fluids and jelly, after which milk foods were allowed as well as cereals. Both of us, and the nursing staff, became convinced that the use of milk in no way prejudiced the patient's recovery. Evidence of fat intolerance was never seen in either group of patients; but starch was not infrequently present in the stool.

The intravenous administration of saline solution was never necessary; bowel wash-outs were never used and dysenteric antiserum was never given. Opium, bismuth and kaolin were used in a few cases, with little faith and with indifferent results. In the period under review sulphaguanidine was at first available only in limited quantities and was kept in reserve for the desperately ill, who, however, failed to appear. It was then reserved for patients with Shiga infections or for those with Flexner infections whose condition did not improve within three weeks. Later, when a more plentiful supply was available, its use was extended. The results have been uniformly good, but it is impossible to assess their significance in such cases as we have seen, many of which tend to rapid spontaneous cure. Nor is our number of persistent cases in which sulphaguanidine was used large enough to enable us to express any definite opinion on its usefulness. Our impression is that it is a valuable drug in the treatment of bacillary dysentery.

Sigmoidoscopy.

A sigmoidoscope was kept in the wards and used frequently in all stages of the disease except the earliest stages. Sigmoidoscopic examinations were carried out in practically all cases in which symptoms persisted for more than ten days. These examinations proved invaluable in prognosis. Changes in the mucosa, usually ulceration, were found in the majority of proved cases in the early stages, and the healing or extension of the lesions was followed in the progress examinations. A description of the various appearances will not be attempted here. It is our emphatic opinion that experience in the use of the sigmoidoscope is essential in the proper management of this disease.

Criteria of Cure.

The criteria of cure which we found satisfactory were the following: (i) the patient feels well and looks well; (ii) the diarrhœa has ceased; (iii) the stools are normal on inspection; (iv) no inflammatory exudate is found on microscopic examination; (v) no abdominal tenderness is present; (vi) the sigmoidoscopic appearances are normal. All these criteria should be satisfied prior to the patient's discharge from hospital. Failure of the stools to yield dysentery bacilli on attempted culture prior to the patient's discharge from hospital was not insisted on. As we have already stated, search for dysentery bacilli in a stool which is normal macroscopically and microscopically involves a waste of time and material.

Discussion.

The foregoing does not purport to be a comprehensive description of bacillary dysentery, but only of the types of cases seen in one hospital in the Middle East during the spring and summer of 1941. The outstanding feature of this group of cases was their mildness. Not only did no death occur, but no patient was put on the "seriously ill" list. Intravenous fluid replacement was never necessary. Post-dysenteric arthritis was uncommon. Iridocyclitis was not seen. We know that deaths from bacillary dysentery have occurred in the Middle East in this period, though we are aware of only one Australian who died. But we realize that our sample, though large in numbers, is only a relatively small percentage of the total number of cases which occurred. We saw 13 Shiga infections; had we seen 100 no doubt we should have observed complicated or fatal cases, but the incidence of such severe cases in the Australian Imperial Force as a whole has been almost negligible. Nevertheless the disease has been responsible for an enormous wastage, as is shown by the fact that the average stay of a patient in hospital in these wards was about one month. This period may seem excessive for a patient with what is after all a comparatively mild illness. But it was our opinion that premature discharge risked not only a relapse for the patient, but the establish-

ment of another possible source of infection among healthy troops. We have already given reasons for our belief that culture of stools prior to the patient's discharge from hospital cannot be relied upon as a means of detecting potential "carriers". It seemed to us better to rely on the vast experience of Colonel Boyd and his colleagues of the Royal Army Medical Corps in India, who have found in many tens of thousands of stool cultures that the healthy subject passing normal stools never excretes dysentery bacilli. For that reason our criteria of cure were fairly rigid, and a patient was not discharged until observation for some days showed that he was free of symptoms and that his stools were of normal consistency and contained no mucus. Probably the introduction of sulphaguanidine will considerably shorten this long stay in hospital.

Acknowledgements.

Throughout we have had the assistance and advice of others with more experience in this field. The design of the dysentery wards and their effective staffing were due to Lieutenant-Colonel E. Britten Jones, late officer commanding the medical division of the hospital. The cooperation of Colonel N. Hamilton Fairley, Consultant Physician, Australian Imperial Force, and the technical advice and provision of serum by Colonel J. S. K. Boyd, Director of Pathology, Middle East, have been invaluable. Major-General S. R. Burston, Director-General of Medical Services, has kindly given permission to publish this paper.

RELAPSING FEVER IN TOBRUK.

BY THE LATE ERIC L. COOPER,

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EARLY in May, 1941, spirochætes were found in the blood of two of the personnel of an Australian Imperial Force field ambulance who were living in a cave in the Tobruk fortress area. During the summer months of 1941 (May until August), 68 patients suffering from relapsing fever passed through an Australian General Hospital in Tobruk. Eight of these were evacuated from the fortress area and were later proved in base hospitals to have been suffering from relapsing fever, although spirochætes were not found in their blood while they were in hospital in Tobruk. The records of 63 of these men have been followed through hospitals and convalescent depots and are sufficiently complete to formulate a clinical picture of relapsing fever in Tobruk.

The majority (39 out of 63) of soldiers affected had been living in caves or in old dugouts constructed by Italian troops. These shelters had not been inhabited by Italians or Libyans for the three months prior to the first appearance of relapsing fever in Tobruk in 1941. Caves, dugouts, tank traps and trenches were infested with rodents. Ticks were found in slit trenches constructed a few weeks previously. One hospital nursing orderly, who had lived in a new slit trench in the beach section of the hospital and who had never been in caves or old dugouts, developed relapsing fever. Relapsing fever in Tobruk was not necessarily acquired in caves, but occurred in men from 27 different units living in various quarters scattered throughout the fortress area.

Some men (19)[1] gave a definite history of having been bitten by ticks, and indubitable tick bites were seen by a medical officer on half (10) of these. Ticks were frequently removed from the skin of soldiers in Tobruk, but not every man bitten by ticks, from an area known to be infested, necessarily developed relapsing fever.

The incubation period of the disease in Tobruk was determined in a few cases in which ticks had been removed from the skin and eight days later the first pyrexial attack began. Although no statistics can be produced, the impression gained was that the more tick bites the soldier suffered, the more likely he was to develop relapsing fever. The total number of relapses occurring during the whole period of the illness may also bear some relation to the number of tick bites.

Clinical Aspects of Tick-Borne Relapsing Fever.

The majority of patients were admitted to hospital with a diagnosis of "pyrexia of unknown origin" ("P.U.O." or "N.Y.D." pyrexia). In some, fever and the presence of spirochætes in a thick blood smear were the only evidence of relapsing fever. The febrile illness was of sudden onset and was associated with headache, anorexia, sweating and the usual concomitants of fever in no way different from the hundreds of cases of "P.U.O." seen in Tobruk.

The headache (58) of patients suffering from relapsing fever was intense, localized behind the eyes or the occipital region. The eyes were sometimes "sore" and had a suffused appearance in some instances. The sweats were often severe (39) and left the patient with a sense of extreme bodily weakness (34) lasting some days after the subsidence of the pyrexia. Appreciable loss of weight (18) during a relatively brief period of pyrexia was a striking feature. After successive attacks of pyrexia the patient became more and more pale with an icteric tint to the skin and conjuctiva; in the most severe cases the patient presented a cachectic appearance within a few weeks.

Pain in the limbs (34) and joints (14) was more severe than in other pyrexial illnesses in Tobruk. Lumbar backache was a major symptom in many (27) cases and recurred with each attack of pyrexia.

In the majority of cases physical examination revealed no localizing signs. A skin rash was occasionally recorded; this was petechial (five) or papular (nine) in character, and difficulty was encountered in deciding whether the rash was due to relapsing fever or was the result of flea bites, which were universal in Tobruk. In some cases (seven) the sites of tick bites were obvious; these had a hæmorrhagic centre with elevation of the skin around it. Bites were most common on the thigh and on the lower part of the trunk. In some cases swelling around these bites recurred with each pyrexial attack, and in a few instances (two) the lymphatic glands draining the bitten area were appreciably enlarged. Usually the mark of a tick bite fades within three days; the presence of an inflamed tick bite on a febrile patient suggests a recurrence of local inflammation due to relapsing fever rather than primary irritation from a bite.

The spleen was palpably enlarged in one-third (19) of the cases, but was recorded as very large in only two instances; usually the edge of the spleen was tender and could just be felt on inspiration. Tenderness in the splenic area in the absence of a palpable spleen was occasionally recorded. The liver was enlarged in few patients (eight); liver tenderness (five) and jaundice (five) were rare.

The lymphatic glands were abnormally large in one-third of the cases (18); the axillary glands (12) were involved more frequently than the cervical chain (six). The lymphatic glands were moderately enlarged, firm, mobile and often slightly tender. No enlargement of glands was regarded as significant if a local cause was present, such as an infected abrasion or ecthymatous sores. Leucocyte counts were made in very few instances, but in two monocyte proportions of 14% and 16% respectively were recorded.

Abdominal symptoms were common; nausea and vomiting often recurred at the onset of each attack of pyrexia. In a few cases vomiting was of severe character. Diarrhœa was not uncommon, and in some instances a bowel infection may have coincided with a pyrexial attack. In some cases, however, diarrhœa recurred with each relapse. Abdominal pain, often severe, was common (21); in some it was referred to the upper or lower part of the abdomen and in others it was of general character. The association of abdominal pain, nausea and vomiting with local tenderness (seven) made diagnosis difficult in some cases.

Frequency of micturition with scalding was not uncommon (seven); examination of the urine revealed no

[1] Where a number appears in parentheses, it refers to the total number of patients presenting the sign *et cetera* under discussion.

pus cells or organisms. Pain in the loin (six) radiating to the groin, testis or penis was often associated with frequency of micturition.

Urinary tract irritation without change in the urine is an interesting feature in this group of patients. The cause has not been discovered.

Chest symptoms and signs were found at a time when respiratory tract infections were unusual in Tobruk. Cough, associated with pain in the chest of pleural character, occurred in one-fifth (13) of the patients. The cough was of an irritating nature with no sputum; hæmoptysis was not recorded. Examination of the chest revealed numerous rhonchi scattered throughout the chest (14); in a few instances râles were also present. The clinical picture was that of acute bronchitis or bronchial congestion rather than of consolidation. In four cases, however, a diagnosis of lobar pneumonia was made; in all of these dulness to percussion, diminished air entry and râles were noted. The physical signs cleared rapidly; this suggested that the pathological basis was the presence of local areas of collapse rather than of infarction or consolidation. Sulphapyridine was given to these patients, and when the temperature fell to normal after two or three days the natural termination of a relapse was attributed to the effect of sulphapyridine or pneumonia. The diagnosis was revised on recurrence of the pyrexia and the discovery of spirochætes in the thick blood film.

Involvement of the central nervous system was not suspected in the earlier cases; but intense headache, pain in the occipital region and neck stiffness were prominent features in many cases. As the headache recurred with each attack of pyrexia and frequently persisted for many weeks after the patient was apyrexial and had returned to duty, it would appear that invasion of the central nervous system is more common than is generally recognized. The future course of these patients who have had a spirochætal infection of the cerebral meningo-vascular system will be of interest both to clinicians and to pension authorities.

One-fifth (13) of the patients had severe neck stiffness at some stage of their illness; some in addition presented Kernig's sign.

The cerebro-spinal fluid when examined (nine) was abnormal in all but two instances. The pressure was usually raised, and the amount of both cells and protein present was increased. The highest number of cells recorded was 540 lymphocytes per cubic millimetre and the highest level of protein was 240 milligrammes per 100 cubic centimetres. A cell content of 60 to 100 per cubic millimetre is a common finding, while the level of protein in the cerebro-spinal fluid is usually raised.

Facial palsy occurred in seven of the 68 patients examined in Tobruk. In every instance this developed late in the disease, many weeks after the patient had been evacuated from the fortress area. In no instance did facial paralysis develop earlier than eight weeks after the onset of the disease. Left-sided facial palsy was more common than palsy on the right side. Bilateral facial palsy occurred; in every instance the paralysis slowly but completely recovered over a period of weeks or months. Neuralgic pain in the facial area was common and was often associated with subjective changes in sensation. Objective sensory loss was rare, and the distribution of sensory abnormality was more often that of the seventh cranial nerve than that of the fifth. Persistent neuralgic pain in other areas was not uncommon. Changes in the cerebro-spinal fluid coincided with the onset of facial paralysis. The cerebro-spinal fluid of two patients, which was normal four and six weeks respectively after the first pyrexial attack, later contained an increased amount of cells and protein when facial paralysis developed.

No "typical" clinical picture of spirochætal relapsing fever in Tobruk can be recorded; a severe febrile illness lasting for a few days with a tendency to recur, associated with spirochætes in the circulating blood, is the only description common to all cases. The fever usually lasted three days—the longest duration of fever in a pyrexial attack was eleven days; other patients with proved relapsing fever had pyrexial attacks lasting only one day. The number of attacks of fever varied greatly; the maximum number of relapses so far recorded for one patient is six. However, some patients are still in hospital, and no accurate statement as to the total duration of spirochætal relapsing fever in this series of patients can yet be made. The mean period spent in hospital by these patients was twelve weeks; one patient has already been under treatment for twenty-six weeks.

Many patients had several pyrexial attacks before they were admitted to hospital; one patient had five "fevers" before spirochætes were found in his blood. The longest period of illness before a diagnosis of relapsing fever was made by the discovery of spirochætes in the blood was twenty weeks. However, patients are still reporting with vague illnesses, and spirochætes are found in their blood. A facial palsy five months after he has left Tobruk may be the first evidence that a soldier has been infected by spirochætes of relapsing fever.

Diagnosis.

The diagnosis of relapsing fever must rest upon the discovery of spirochætes in the circulating blood. This diagnosis is never easy. The parasites are few in number and are best seen in a "thick" preparation. Even in the hands of a pathologist experienced in this diagnosis, smear examinations may have to be repeated many times before spirochætes are found. In 16 cases in this series blood films were examined with negative results on one or more occasions before spirochætes were finally detected. The best time to find parasites is at the beginning of a febrile attack. It rarely happens that a soldier is admitted to hospital in the first twenty-four hours of a pyrexial illness. If relapsing fever is suspected the patient must be retained in hospital until a further attack of pyrexia begins. Even though the blood was examined during a "fever" on 17 occasions, negative results were obtained in cases in which "positive" films were later obtained. In some cases as many as three "negative" films were examined before a "positive" was obtained. Even patients known to be suffering from relapsing fever, may provide a "negative" smear during a relapse. On the contrary, it is always worth while to make a smear examination in an afebrile period, as spirochætes were found in the blood of 22 patients at times when the temperature was normal. To sum up, the diagnosis of relapsing fever is never easy; there is no single clinical picture; spirochætes must be sought over and over again during fever and during afebrile periods. Relapsing fever must be suspected in every case of fever, if the patient has been in an area known to be infested with ticks carrying spirochætes.

Treatment.

There is no specific treatment for spirochætal relapsing fever. Arsenic was given intravenously to the majority of the patients in this series. The total quantity of arsenic given to all but four patients was inadequate. Arsenic is a specific louse-borne relapsing fever, but in similar doses has no effect on the tick-borne variety. Spirochætes were found in the blood of 17 patients in this series who had previously been given intravenous injections of arsenic. A pyrexial relapse occurred in 41 cases after the administration of arsenic. However, the effect of arsenic in this disease must be regarded as still not proved. The doses of arsenic used seem inadequate for the treatment of a spirochætal disease in which there is evidence of invasion of the central nervous system and the reticulo-endothelial tissues. Compared with the dosage of arsenic used in syphilis, the quantities of arsenic administered to patients in this series was not sufficient to enable an opinion to be expressed as to its efficiency.

Summary.

1. The records of 68 patients seen in a Australian general hospital in Tobruk between May and August, 1941, have been reviewed.

2. Fever associated with the presence of spirochætes in the blood smear is the only feature common to all these cases. Some patients had in addition symptoms and signs suggesting involvement of the central nervous system, the reticulo-endothelial tissues, the renal tract or the lungs.

3 Late involvement of the central nervous system with facial paralysis is relatively common.

4. Tick-borne relapsing fever does not respond to arsenic given intravenously in the doses used for treatment of louse-borne relapsing fever.

Acknowledgements.

Major-General S. R. Burston, Director-General of Medical Services, has kindly given me permission to publish this paper.

Appendix.

The following is a summary of the conditions and findings in cases of relapsing fever in Tobruk.

General.

Total patients with relapsing fever in an Australian General Hospital in Tobruk	68
Diagnosis of relapsing fever not made in Tobruk, but spirochætes found in blood at base hospitals	8
Records sufficiently complete for purpose of this paper	63
Ages varied between 46 years and 21 years (over 35 years 9 patients).	
Units from which patients were admitted numbered 27.	
The largest number from any one unit, 13.	
Four infantry battalions produced 32 cases out of the total 68.	

Quarters.

(Conditions recorded in 53 cases.)

Living in caves	29
Living in old dugouts previously occupied by Italians	10
Living in new dugouts or slit trenches recently constructed	14

Relation to Tick Bite.

(Number recorded, 55.)

Nineteen men bitten by ticks—	
Patients reported having been bitten by ticks	9
Tick bites seen by a medical officer	10
Ticks seen in quarters	11
Others in unit affected, but patient gave no history of tick bite or ticks in his quarters	13
Patients stated that no ticks had been reported in his quarters	12

Symptoms.

(a) General.	
Headache, severe	58
Vertigo	15
Insomnia	7
Backache, severe	28
Pain in limbs	34
Marked loss of weight during pyrexial attacks	18
Pain in joints	14
Sense of general weakness	34
Anorexia	22
Shivers	19
Sweats, severe	39
(b) Abdominal.	
Abdominal pain	21
Nausea	21
Abdominal tenderness	7
Vomiting	13
Diarrhœa	7
(c) Urinary.	
Frequency of micturition	9
Scalding on micturition	7
Pain in loins radiating to penis or testicles	6
(d) Respiratory.	
Cough	13
Pain in chest	7

Physical Signs.

Spleen palpable	19
Spleen tender	6
Tenderness over spleen area, but spleen not palpable	5
Liver enlarged	8
Liver tender	5
Jaundice	5
Marked pallor of skin	14
Skin rash	14
Lymphatic glands enlarged in cervical region	6
Axillary glands enlarged (without local cause)	12
Rhonchi in chest	14
Râles in lungs	5
Dulness to percussion and diminished air entry at the base	4
Neck stiffness	13
Kernig's sign present	4
Facial paralysis	7
Cerebro-spinal fluid examined	9
No abnormality in cerebro-spinal fluid	2
Highest cell counts	540
Lowest cell counts	20
Highest cerebro-spinal protein, milligrammes per 100 cubic centimetres	240
Lowest cerebro-spinal protein, milligrammes per 100 cubic centimetres	25

Fever.

Highest temperature recorded, 105° F.
Temperature over 103° F. in 23 patients.
Longest duration of fever in any one pyrexial attack, 11 days.
Shortest duration of fever, 1 day.
Total number of pyrexial attacks in any one case varied between 1 and 10 attacks, the mean number 4 attacks.
Total duration of illness before return to duty varied from 3 to 26 weeks, the mean being 12 weeks.[1]
The largest number of pyrexial attacks before the patient was admitted to hospital was 5; the mean number of attacks before admission was 2.

Diagnosis.

The longest period of illness before spirochætes were found in the blood was 20 weeks in one case.
The blood was examined and no spirochætes were found, although later blood smears gave positive results, in 16 cases.
Although the patient was febrile, the blood smear gave negative results in 17 examinations.
The maximum number of "negative" smears before a "positive" was obtained was 3.
Spirochætes were found in blood smears taken while the patient's temperature was normal in 22 cases.
Patient had a febrile attack after diagnosis had been made, yet no spirochætes could be found in thick blood smears, in 15 attacks of pyrexia.

Treatment.

Arsenic given intravenously	57 patients
One injection of an organic arsenical given	25 patients
Two injections of arsenic given	17 patients
Three or more injections given	15 patients
Maximum arsenic given	3·45 grammes
Over two grammes arsenic given	4 patients
Over 1·5 grammes arsenic given	14 patients

Mean period after first attack of pyrexia when arsenic was first given was 3 weeks.
A relapse occurred after arsenic was given in 41 cases.
Although arsenic had previously been given, pyrexia recurred and spirochætes were found in thick blood smears of 17 patients.

Reports of Cases.

MACROCYTIC ANÆMIA OCCURRING IN A CASE OF INFECTIVE HEPATITIS.

By J. M. BONNIN,

Captain, Australian Army Medical Corps, Abroad.

Clinical Record.

CORPORAL W.C., aged twenty-two years, of the Royal Australian Air Force, was admitted to an Australian General Hospital on October 5, 1941. He was one of a number of patients suffering from infective hepatitis arriving from the Western Desert of Egypt. He had always been in good health, except for an attack of diarrhœa which lasted for one day only, and which he had contracted in Tobruk one year previously. On admission to hospital he gave a history of the sudden onset of backache, sore eyes and a temperature of 102° F. ten days previously, and of having become jaundiced three days later. He had had a shivering attack on the day of his admission to hospital, but not before.

[1] Some men are still in hospital.

On examination he was deeply jaundiced to a bright orange-yellow colour. The liver was tender and enlarged to one inch below the costal margin. The spleen was palpable and slightly tender and the temperature was 101° F. The urine contained bile pigment. He was given a light fat-free diet, with two drachms of magnesium sulphate in the mornings. Three days later he was still pyrexial; the spleen was firmer and palpable one inch below the costal margin. In view of this condition blood smears were examined, but no malarial parasites were seen. The stools were examined for the first time on this day and were found to be clay-coloured. A week later his condition was unchanged, though he developed severe pruritus. The blood coagulation time was then three and a half minutes, which is the upper limit of normal by the method used.

On October 24 a blood examination gave the following information. The hæmoglobin value was 85%,[1] the red blood cells numbered 3,730,000 per cubic millimetre, the colour index was 1·1; the white blood cells numbered 6,000 per cubic millimetre; a differential white cell count showed that 71% were neutrophile cells, 22% were lymphocytes, 7% were monocytes, and no eosinophile cells were present. One week later the urine contained a large quantity of bile pigment and salts, with numerous bile pigment casts, but no albumin. A duodenal tube was passed and two drachms of magnesium sulphate in 30 cubic centimetres of water were introduced. In five minutes ten cubic centimetres of slightly bile-stained fluid were withdrawn and inoculated into nutrient media. A few colonies of *Staphylococcus aureus* were obtained and regarded as of no significance. The patient's general condition remained good. He was bright and cheerful, he ate well and slept well in spite of the pruritus, and he did not appear to have lost weight, though his temperature still rose to 100·5° F. in the afternoons.

On October 31 (five weeks after the onset of the disease) he was given a blood transfusion of 400 cubic centimetres from a compatible donor, convalescent from infective hepatitis, as a therapeutic measure. This was followed by a mild rigor with a rise of temperature to 104° F. On November 2 the blood picture was as follows: hæmoglobin value 70%, red blood cells 2,200,000 per cubic millimetre, colour index 1·6. Examination of the blood film revealed macrocytosis with some anisocytosis, but facilities for estimating the size and volume of the corpuscles were not available. From then on the blood was examined at frequent intervals and the results can be seen from the accompanying chart (Figure I).

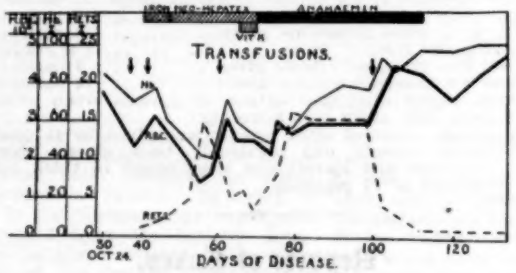

FIGURE I.

Graph showing blood changes from the time of onset of anæmia to the time of recovery. It illustrates the rapid increase of anæmia in spite of two blood transfusions and iron therapy, its arrest by liver therapy, and its response to a large blood transfusion. This is followed by an increase in severity, then by a definite response to liver therapy, and by a further response to the fourth transfusion. It can be seen that following the cessation of liver therapy the anæmia, though mild in degree, reverted to the macrocytic type, as demonstrated by the divergence of the graphs of hæmoglobin and red blood cells. This is finally followed by a return to normal.

On November 4 the patient was again given a transfusion of 400 cubic centimetres of blood from a patient convalescent from infective hepatitis. By this time jaundice was intense, but the bright orange-yellow colour had changed to an olive-greenish yellow; the spleen was slightly larger, as was the liver, but the latter had lost its acute tenderness. The stools remained pale. In view of the continued pyrexia a blood culture was attempted, but no organisms were recovered. Liver extract in the form of "Neo-hepatex" was administered intramuscularly in doses of two cubic centimetres per day; the dosage was increased to six cubic centimetres two days later, on the advice of Colonel Hamilton Fairley. On the eighth day of liver therapy a reticulocyte response of 14% was noted, but the hæmoglobin value and the number of red blood cells did not alter appreciably. However, the preceding continuous drop in both was arrested, as can be seen by reference to Figure II, in which are shown in greater detail the blood changes during this critical stage of the disease. On the day of the maximum reticulocytosis the hæmoglobin value was 42% and the red blood cells numbered 1,400,000 per cubic millimetre.

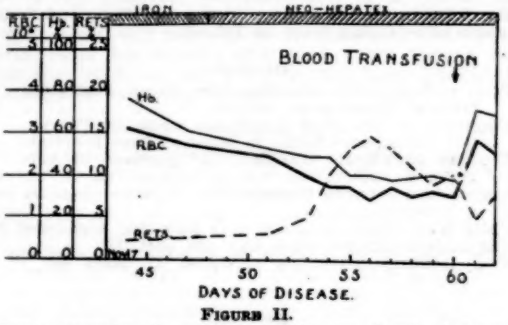

FIGURE II.

Graph showing in greater detail the blood changes during the first reticulocyte response to liver therapy.

In spite of this arrest of the previously progressive anæmia, his general condition began to deteriorate rapidly. "Neo-hepatex" therapy then had to be reduced to one cubic centimetre per day, owing to shortage of supply. The temperature rises became higher and more irregular, while the pulse rate, which had previously been relatively slow, increased. The patient became thinner and abdominal distension developed. An apical systolic murmur made its appearance. By November 20 the blood coagulation time had risen to eight minutes. Examination of a blood film revealed macrocytosis with some anisocytosis and slight poikilocytosis. The mean corpuscular diameter was 8·8μ. The patient became drowsy, the jaundice became more green than yellow in colour, and his condition became so serious that he was placed on the "dangerously ill" list. The temperature chart during this period is reproduced in Figure III. He was then given a large transfusion of 2,100 cubic centimetres of blood over a period of fourteen hours. This caused a considerable improvement in the blood picture, the hæmoglobin value rising from 38% to 72%. Prior to this transfusion the consensus of opinion was that the prognosis was hopeless. Following the transfusion, though he was still seriously ill and somewhat drowsy for a further two days, his condition appreciably improved, so that some hope of his ultimate recovery could be entertained. Treatment with vitamin *K*, in the form of daily injections of one cubic centimetre of "Prokayvit", each cubic centimetre of which contains five milligrammes of 2-methyl-1:4 naphthoquinone in oily solution, was commenced on November 29 and continued for four days. At this stage of the disease the stools were noticed to be regaining their normal colour, having been pale for eight weeks. On December 4 the liver extract was changed to "Anahæmin", two cubic centimetres per day being given. On December 8 Van den Bergh's test produced a positive biphasic reaction, the serum containing eight Van den Bergh units of bilirubin, equivalent to four milligrammes of bilirubin per 100 cubic centimetres. On December 12, eight days after the commencement of "Anahæmin" therapy, a second reticulocyte response of 16% was noted. On this day the hæmoglobin value was 57% and the icteric index was 70.

From then on the improvement in the patient's general condition was progressive, despite fluctuations in the blood count; the jaundice began to fade and he began to gain in weight and to lose his abdominal distension. The liver and spleen remained enlarged, but lost all tenderness. The hæmoglobin value continued to rise and the icteric index to fall. A fractional test meal examination performed on December 29 revealed only a trace of free hydrochloric acid. By January 2, 1942, the blood picture had remained stationary for a week, the hæmoglobin value being 76%, so another blood transfusion was decided upon and 1,200 cubic centimetres of blood were given. Twelve days later the hæmoglobin value was 90%, the red blood cells numbered 4,360,000 per cubic millimetre and the colour index was 1·0, while the icteric index had fallen to 17. The patient was now in

[1] All hæmoglobin readings were made with a Sahli hæmoglobinometer, the same instrument being used each time; 100% on this Sahli scale is equivalent to 15·5 grammes of hæmoglobin per 100 cubic centimetres, which is taken as the average reading for the normal male adult.

excellent condition and had only a faintly icteric tint. He felt perfectly fit; he was eating a full diet, was sleeping well and was up and about all day. Liver therapy was discontinued. One week later he still had a faint icteric tint, his liver was reduced in size, the spleen was palpable half an inch below the left costal margin, and his good general condition was maintained. The blood picture had, however, changed and was returning towards the macrocytic type. The findings were as follows: hæmoglobin value 95%, red blood cells 3,500,000 per cubic millimetre, colour index 1·3, reticulocytes 1%. Examination of a film revealed macrocytosis with slight anisocytosis. The mean corpuscular diameter was now 8·4μ. The next week, however, produced a partial return to normal; the hæmoglobin value was 100%, the red blood cells numbered 4,200,000 per cubic millimetre, and the colour index was 1·2. By now Gamelin's test for bile pigment in the urine produced no reaction. No excess of urobilinogen was demonstrated by Ehrlich's aldehyde reagent. The fractional test meal examination, repeated on January 23, revealed a normal acid curve, the highest free acid value being 48, expressed as cubic centimetres of N/10 caustic soda per 100 cubic centimetres; this occurred at the end of one and a half hours. The blood serum did not agglutinate suspensions of *Bacillus typhosus*, *Bacillus paratyphosus* A, B or C, or *Brucella melitensis* or *Brucella abortus* in dilutions greater than one in twenty-five. Repeated examinations of the stools for amœbæ or ova gave negative results. Attempted culture of the stools yielded no growth of pathogenic organisms. The Kahn test produced no reaction. Two weeks later, when the patient was last examined, he had made a complete recovery, apart from a slight icteric tint of the conjunctivæ. The liver was just palpable at the costal margin, while the spleen was no longer palpable. The blood picture had returned to normal. On no occasion had nucleated red blood cells or abnormal white blood cells been seen in stained films.

Discussion.

The Diagnosis.

This case was considered to be one of infective hepatitis, one of an epidemic that occurred in the Western Desert at this time. The majority of the cases in this epidemic differed in no essentials from those seen in previous epidemics, both in the Middle East and in Australia. Amongst other cases of infective hepatitis in hospital at this time were two more in which the course was considerably longer than the average. In both there was evidence of complete biliary suppression or obstruction, in that the stools were clay-coloured for prolonged periods. One of the two patients developed hæmorrhages, subcutaneously and from the mucosa of the nose and rectum, which responded to the administration of vitamin *K* and blood transfusions; but neither developed a macrocytic anæmia. On the other hand, there were other patients with the disease in so mild a form that they "reported sick" only on being ordered to do so because of the icteric tint of their conjunctivæ. Between these two extremes there were many intermediate degrees of the disease, ranging from one extreme to the other. One unusual feature of this present case was the prolonged irregular fever, which persisted for twelve weeks. Figure III is a specimen of the temperature chart during the period when the range of temperature in irregularity and degree was at its greatest and the patient was most gravely ill. As no other cause could be found, this prolongation of fever was attributed to a persistence of the disease process in the liver. An attempt to combat this process was made by the administration of two transfusions from subjects convalescent from infective hepatitis. That this treatment had no beneficial effect, apart from the transitory improvement in the blood picture, was shown by the continued downward course of the disease. It is in fact a possibility that instead of benefiting from these transfusions the patient's condition became worse. On the complete temperature chart, which unfortunately cannot be reproduced in detail, because of its length, it can be seen that the temperature became considerably higher and remained so for five days (the fourteenth to the eighteenth) after the convalescent blood transfusion. A similar occurrence followed the second transfusion. The consensus of opinion is that epidemic infective hepatitis is a virus disease, though proof is lacking, mainly because a susceptible laboratory animal has not yet been found. As both of these donors came from a different area (namely, Syria), the possibility that new strains of virus had been introduced was considered. That jaundice, probably identical with epidemic infective hepatitis, can be produced by intravenous serum therapy was pointed out by Findlay and McCallum,[3] who reported the occurrence of cases of the disease following the use of pooled convalescent serum in the prophylaxis of other diseases, though in their cases jaundice did not appear for two or three months after the administration of serum. However, in this case it is most probable that these exacerbations of fever were manifestations of the original disease and not due to the transfused blood, though why the patient should have remained febrile for so long cannot be explained. *Spirochætosis ictero-hæmorrhagica* was excluded by the absence of this disease in the Western Desert.

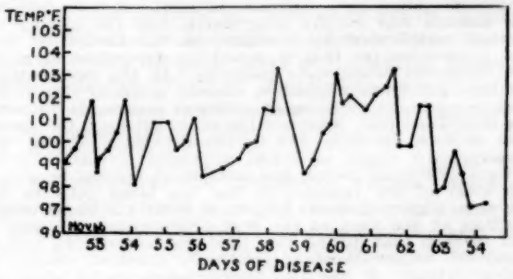

FIGURE III.

Specimen of the temperature chart showing the irregularity and degree of fever over a twelve-day period when the patient was most seriously ill.

The Stools.

As in previous epidemics, the transitory appearance of clay-coloured stools was usual. The explanation for this is that the inflamed liver cells fail to secrete bile in the normal amount. In addition, these swollen cells exert pressure on the smaller biliary vessels. Recent work by van Rooyen,[2] who examined the centrifuged deposit from bile in early cases, revealed no evidence whatever that the inflammatory process extended to the biliary vessel walls. In the single subject he examined *post mortem* the predominant pathological lesion evident was liver cell necrosis of the midzonal type. There was no evidence of obstruction of the bile ducts. In one of the other two cases referred to earlier the stools remained pale and the patient jaundiced for six weeks after the fever, which was of short duration only, had subsided. This observation has frequently been reported before. In such cases it would seem that the recovery of the function of the damaged liver cells to excrete bile is unusually prolonged, though the presence of mechanical obstruction of larger bile vessels must be considered as a possibility.

The Anæmia.

The anæmia was considered to be caused by a deficiency of the pernicious anæmia factor complicated by some other factor, possibly excessive hæmolysis. That extensive disease of the liver can cause macrocytic anæmia is well recognized. Van Duyn[2] reported its occurrence in several types of liver disease, including five out of 28 cases of cirrhosis and in 12 of 30 cases of poisoning with arsenuretted hydrogen. The mechanism whereby this occurs is not fully understood. One theory is that a sufficiently diseased liver cannot store the "P.A. factor" for use by the bone marrow as required. In this case there is ample clinical evidence that the liver was grossly damaged, as it had been the site of acute disease for one month prior to the onset of the anæmia. Wintrobe and Shumacker[4] reported a comparable case of macrocytic anæmia occurring in catarrhal jaundice, though in their case the duration of the disease was considerably shorter than in the present case. The view that this anæmia was primarily due to the liver disease is also supported by the temporary recurrence of macrocytic anæmia immediately after liver therapy was suspended, and this in spite of the fact that the patient had had daily injections of two cubic centimetres of "Anahæmin" until ten days previously. Had the liver had its full capacity to store the "P.A. factor", as it has in Addisonian pernicious anæmia, then there should have been sufficient "P.A. factor" in the body to prevent the recurrence of anæmia for several weeks. This fact also suggests that the damage suffered by the liver as regards its erythropoietic function had not fully recovered by the time that liver therapy was discontinued.

The tendency towards macrocytosis is well demonstrated in Figure I, in which will be noticed the constant tendency for the graphs of the hæmoglobin and red blood cells to diverge. It will also be noticed that this diversion is corrected by all transfusions by which dilution with normal blood has brought the total blood picture towards normality. The same effect is observed following treatment with liver, more especially during the second reticulocyte response, in which the means of dilution of the macrocytic blood is, as

it were, with reticulocytes. Minot and Castle[2] pointed out that in uncomplicated pernicious anæmia treated with adequate dosage of liver the reticulocyte response obtained bears a relation to the original red cell count. Furthermore, if this relation is not obtained, then the diagnosis must be reconsidered. When the original count is 2,700,000 per cubic millimetre, as it was in this case, the anticipated maximum reticulocyte response is 10%. But in this case the anæmia was rapidly progressive, and the picture was further complicated by toxæmia, as manifested by fever and prostration, so that it would be impossible to predict the maximum reticulocyte response. In the circumstances the response actually attained, namely, a steady rise to 15%, may be regarded as being of sufficient magnitude to justify the conclusion that, whatever the exact ætiology, the anæmia was at least partially due to the deficiency of the "P.A. factor".

Some additional factor was considered present because of the form of the response of the red blood cells to liver therapy. Figure II shows in greater detail the hæmatological findings at the time of the first reticulocyte response. It will be seen that during this time the hæmoglobin and red blood cell values remained practically constant, despite the reticulocytosis. It has also been pointed out by Minot and Castle[2] that during the course of treatment of Addisonian pernicious anæmia an intercurrent fever will nullify the response to liver therapy. In the graph from their case they demonstrate that the number of red blood cells can be seen to rise and fall with that of the reticulocytes. In this present case, as has been pointed out, this did not occur, and it is this fact that made one consider another factor to be present. As the reticulocytosis denies the existence of any gross dyshæmopoiesis, this other factor is presumably abnormally rapid hæmolysis. This may be thought to have been the result of the rapid destruction of the red blood cells that the patient had received in the first two transfusions. However, these transfusions were given sixteen and twelve days respectively before the onset of reticulocyte response, so that if these transfused cells were hæmolysed more rapidly than normal, then the majority of them would have been already destroyed. Consequently these transfusions could not influence the blood picture to any great extent at this stage. Then again, at the time of the second reticulocyte response the hæmoglobin and red blood cell levels rose concurrently for a few days; this occurrence resembled more closely the type of response seen in Addisonian pernicious anæmia. It was noticed that this occurred only when the fever had temporarily almost subsided, and when the fever again increased the improvement in the red blood cell level was arrested, although a high reticulocyte percentage was maintained. This shows that in this recrudescence of the disease some other factor, presumably hæmolysis, was again introduced to delay the rise in the number of red blood cells despite the sustained reticulocytosis. It should be made clear, however, that the response of the macrocytic anæmia of liver disease to liver therapy does not necessarily, or indeed usually, follow the classical type of response seen in pernicious anæmia. It seems that the liver may be involved in erythropoiesis in some more obscure manner than in the purely mechanical one of storing "P.A. factor". When this function of the liver has been more fully elucidated, then this postulation of a concurrent hæmolytic process may need to be abandoned. It has been introduced in a speculative manner without any absolute evidence of its existence, in an attempt to explain some of the phenomena observed. It must be pointed out here that the second reticulocyte response reached its peak on the eighth day after the substitution of a new preparation of liver, and in addition the daily dose was increased from one cubic centimetre to two cubic centimetres. That this had a bearing on the second reticulocyte response was possible; but, as was pointed out earlier, the lowering of the patient's fever at this time may have been the more important factor. After the ninetieth day of disease it was found that the number of red blood cells did not continue to rise as expected, although the patient remained apyrexial and the reticulocyte percentage was maintained. After the number had been stationary at 3,000,000 per cubic millimetre for one week the fourth blood transfusion was given, and this brought the level to 3,700,000 per cubic millimetre; the level then continued to rise, to reach 4,200,000 per cubic millimetre in four days. It then seemed that this was to be the beginning of the final return to a normal blood picture. However, one week later the number of red blood cells had again fallen and the colour index had risen to 1·3. The mean corpuscular diameter was 8·4μ. As was suggested earlier, this return to macrocytosis may have been due to a temporary persistence of lowered liver function in erythropoiesis. If so, this would probably not have occurred had liver therapy been continued a little longer. However, no more liver was administered and the blood picture from then on gradually returned to normal status, which it attained in a further two weeks.

Iron, in the form of ferrous sulphate (three grains three times a day), was administered early in the disease, but it did not affect the downward trend of the anæmia; its effect, if any, was difficult of evaluation owing to simultaneously administered blood transfusions. Moreover, at that time frequent investigations were not carried out, as the onset of severe anæmia was not anticipated. The patient ate well throughout the illness, and as the colour index was consistently greater than 1·0 there is nothing to suggest that iron deficiency played a role in the ætiology of the anæmia that arose later in its course. The hæmatological investigations made in this case are incomplete; estimations of mean corpuscular volumes were obviously desirable, but the necessary equipment was not available in the laboratory.

Summary.

A case of infective hepatitis has been described. Unusual features of the case were the prolonged febrile course with splenomegaly and pale stools, the green colour attained by the jaundice, and the development of a severe macrocytic anæmia which rsponded to liver therapy.

Acknowledgement.

I should like to express my gratitude to Colonel N. Hamilton Fairley for his advice, to Lieutenant-Colonel Ian Wood for his constant assistance, and to Major E. V. Keogh, who with Miss Freeman made all the laboratory investigations.

Major-General S. R. Burston, Director General of Medical Services, has kindly given me permission to publish this record.

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Reviews.

RADIOGRAPHY IN PREGNANCY.

A BOOK entitled "Clinical Roentgenology of Pregnancy", by Dr. William Snow, is a small work, clearly printed and with excellent illustrations; the author is director of radiology at Bronx Hospital and röntgenologist in charge at Harlem Hospital, New York City.[1]

The book is divided into six chapters and a comparatively large section on case reports. The first chapter begins with a general discussion on the use of radiology in pregnancy, the author pointing out that recent advance and modern technique have made for more accurate measurements. The author compares various pelvimetric methods and states that the doses of X rays used for diagnostic purposes are now safe for mother and child.

The second chapter deals in some detail with the placing and radiographing of the patient. The technique is simple, established positions being used.

Chapter III deals with pelvimetry and cephalometry. The author describes his method of making corrections for distortion and uses linear rather than volumetric measurements, as the former are the measurements to which the obstetrician is accustomed. The shape of the pelvis and its measurements are discussed, together with the fœtus and its relationship to the pelvis, abnormalities of position and fœtal deformities. This chapter is well illustrated.

The final chapter deals with the soft tissues in pregnancy, particularly the visualization of the placenta, and the diagnosis of *placenta prævia*, with many good radiographs. The last sixty-one pages are devoted to case reports with radiographs of every case.

This volume is interesting and instructive and should prove valuable to both radiologist and obstetrician.

[1] "Clinical Roentgenology of Pregnancy", by W. Snow, M.D.; 1942. Springfield: Charles C. Thomas. Super royal 8vo, pp. 192, with 119 illustrations and 11 charts. Price: $4.50 net.

The Medical Journal of Australia

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WORK, HEALTH AND EFFICIENCY.

At the present time industry has two main objectives, and only two. In the first place it aims at the production of more and yet more munitions of war, that victory may be wrested speedily from a malignant enemy. Secondly, industry sets itself to produce goods to meet the essential needs of the community. It knows that if the first of its objectives is to be achieved, goods for the everyday needs of the people must be turned out with the least possible delay and by the smallest number of people that can produce them—as many fit persons as possible must be set free for service with the armed forces and for the making of munitions. There can be few persons in the community who are not alive to the need for the organization of industry along lines that will enable it to attain its objectives; few who do not agree that every man and woman should be doing the work for which, either by native ability or as a result of special training, he or she is best suited; few, we hope, who will deny that goods for the essential needs do not include what are the "frills" of everyday existence, the luxuries, the hundred and one extras with which we can all dispense. Yet in spite of all this there are many who hold that industry is not achieving the best results because the human body is being treated as if it were a tireless machine and not flesh and blood activated and controlled by a sensitive nervous system. Those who put forward this view are just as earnest as those patriots who think that they will increase production by keeping men and women at work day after day, week after week, month after month and year after year, in anything up to twelve-hour shifts or more. To support their contention they have scientific observation from many parts of the world supported by unquestionable facts. Ever since this war began, the subject of fatigue in industry and its influence on output has been discussed in medical journals in England and in THE MEDICAL JOURNAL OF AUSTRALIA, but those in control of war industry have shown themselves surprisingly unwilling to accept anything conflicting with their own preconceived notions. In spite of this, or perhaps because of it, the facts regarding work, the human body and industrial output must be made known; some day enlightenment may come.

The occasion of the present reference to the subject is the receipt from England of "Emergency Report, Number 2", published by His Majesty's Stationery Office for the Industrial Health Research Board of the Medical Research Council of the Privy Council.[1] Since we are reemphasizing previously expressed views about industrial fatigue, it will be appropriate to point out once more that the Industrial Health Research Board in Great Britain was formerly known as the Industrial Fatigue Research Board, and that it was appointed in direct succession to the Committee on the Health of Munition Workers set up during the war of 1914-1918. The terms of reference of the board are as follows:

> To suggest problems for investigation, and to advise upon or carry out schemes of research referred to them from time to time by the Medical Research Council, undertaken to promote better knowledge of the relation of methods and conditions of work to functions of the human body, having regard both to the preservation of health among the workers and to industrial efficiency; and to take steps to secure the cooperation of industries in making widely known such results of this research work as are capable of useful application to practical needs.

These terms of reference are quoted so that stress may be laid on the fact that preservation of health among workers is put first and industrial efficiency second. A medical hygienist must always adopt this attitude. In the many industrial undertakings connected with war certain processes may be injurious to health. It is the duty of the medical hygienist to see that ways and means are devised by the use of which the worker can carry out the process without injury to himself. Industry must never be regarded from the point of view of results only—it can never be efficient if the health of the worker is in jeopardy. When, therefore, we find that efficiency can be promoted and production increased by measures which make life tolerable and even perhaps pleasant for the worker, there is, or should be, no room for argument.

The Industrial Health Research Board's present report deals with an investigation covering fifty factories, representing approximately 200,000 workers. It is interesting to note, not only that at first the board had difficulty because its inquiries were restricted to a few government factories, but also that records were often unsuitable or not available at all. Apparently absenteeism during overtime was either neglected in records or very imperfectly recorded. The report is divided into three sections and may with advantage be considered according to this arrangement. Unfortunately a detailed description of the findings cannot be given. In the first section, which has to do with lost time, the available figures were obtained from records which, as already stated, were incomplete—the records usually referred to a normal week of forty-seven hours. The figures for absenteeism in overtime are usually higher than those for normal working periods, and the available figures therefore understated the total loss. When this was taken into consideration it was found that the time lost owing to sickness, injury and absence without permission varied, when there were

[1] "Hours of Work, Lost Time and Labour Wastage", Medical Research Council, Industrial Health Research Board, Emergency Report, Number 2; 1942. London: His Majesty's Stationery Office. Royal 8vo, pp. 30. Price: 6d. net.

no extraneous factors, with the weekly hours of work. The time lost was reasonably low when the hours of work were less than sixty per week, but was greater and in some cases excessive when the hours were from sixty-five to seventy-five a week. During the height of the emergency period in June and July, 1940 (the retreat from Dunkirk took place in May) the increase was mainly due to the fatigue, strain and ill health caused by the longer hours of work, and we are told that "there is little doubt that a continuation of these hours would have had serious and far-reaching effects". The introduction of week-end breaks which became fairly general in August, together with holiday rota schemes, was followed by an improvement. From then on there was little evidence that the hours of work were responsible for more than the normal amount of fatigue. Among the causes of absenteeism that are mentioned are seasonal causes and air raids. Women lost more time than men because of home duties and family responsibilities.

In the section of the report devoted to hours and output results are discussed in two groups according to whether the work is mechanized or whether the output is largely dependent on the worker. Clearly output and hours cannot be considered independently of absenteeism. The board is careful not to claim too much from its observations and states that though the results are not conclusive they give some indication of the nature and effects of factors which increase or retard productive effort. In the statements made about absenteeism the position in regard to length of hours is implied. It could not be expected that the magnificent effort in production made in some factories after the evacuation from Dunkirk could be maintained for long. To do this would have been physiologically impossible. The urgency of the situation provided a stimulus so great, however, that output has in nearly every case investigated remained above "the previous level". We are also told that the output from time to time was stimulated by successes and depressed by defeats. The institution of holidays almost invariably led to an increase in the rate of working. Holidays seemed to dispel the last traces of fatigue and strain caused by long periods of intensive activity, and they appeared to be more effective than the preceding sequence of week-end breaks. Other factors influencing the output of work are mentioned. Long hours of constant work impair the usefulness of machines as well as of men; it was found that less time was lost through defects in machinery when time was available for proper overhaul and repair of machines. When organization of work was improved and methods of working were perfected the output naturally was greater.

To the third portion of the report, that dealing with labour wastage, short reference only is necessary. Some of the conditions leading to a high rate of wastage included the employment of women who were unaccustomed to factory work or of married women whose home responsibilities made it difficult for them to adjust themselves to factory life. In this regard difficulties of shopping, of the preparation of meals and of transport were important. Labour wastage was due largely to workers who left their employment of their own accord; the number dismissed was usually very small.

The report, so far as it goes, confirms previous findings. Whether the tentative suggestion that hours of work over an extended period should not exceed sixty to sixty-five a week for men and fifty-five to sixty for women is correct remains to be seen. If it should prove to be correct for Great Britain it will probably, but not necessarily, be true for Australia. The matter is of such importance to Great Britain that the research workers of the Industrial Health Research Board are to continue their studies. Australia should either accept the findings of this scientific body working in England and adjust the working conditions of its war-time industries, or it should set up an investigation into fatigue as it affects the health of Australian workers in Australian factories and their war-time output.

Current Comment.

THE Rh FACTOR.

WHEN Landsteiner in 1901 first described certain properties of the blood which later on became known as the blood groups, neither he nor his contemporaries could imagine that his discovery would have the far-reaching effects which now, forty years later, are illustrated by every soldier who has his blood group engraved on his identification disk. Practical application of Landsteiner's discovery has been slow, and much confusion has been created by the adoption of two opposite numerical classifications, and although the League of Nations Health Committee officially recognized and recommended only the ABO classification, the old numerical one is still too often used in this country. This practice has a definite disadvantage, as it does not allow for the fact that, instead of one factor A, there are really two factors, A_1 and A_2; that is, instead of the groups A and AB there are actually A_1, A_2, A_1B and A_2B. As A_2 is often very weak, a number of people are still wrongly grouped B instead of A_2B and O instead of A_2. In 1927 Landsteiner and Levine discovered a new series of factors, which were independent of the ABO series. The M and N factors, as they were called, occur either individually or in combination with each other (there is no O group in this series), and with their help the population could then be divided into twelve groups instead of the previous four. As no anti-M or anti-N agglutinins normally occur, these factors have no importance for blood transfusions, but are of great value in legal cases, especially in the establishment of paternity. In 1940 K. Landsteiner and A. S. Wiener[1] reported on a new agglutinable substance of human blood unrelated to the other agglutinogens. This new property has been designated Rh because it was first detected with the aid of an immune serum prepared by injecting the blood of rhesus monkeys into rabbits. It was soon seen that this new discovery was of considerable clinical importance.

Physicians and surgeons have been troubled for some time by transfusion accidents, even in cases in which there was no doubt about the correctness of the blood grouping and matching. It became evident that the patients in whom such accidents occurred fell into two classes. One of them consisted of patients who had to receive a considerable number of blood transfusions at fairly short intervals. The other group consisted of women in whom complications of pregnancy necessitated blood transfusions, and in this group accidents often occurred even at the first transfusion. K. Landsteiner and A. S. Wiener,[2] as well as P. Levine and his collaborators,[3] have made exhaustive studies of such cases,

[1] *Proceedings of the Society of Experimental Biology and Medicine*, February, 1940.

[2] *Annals of Internal Medicine*, June, 1940; *Archives of Pathology*, August, 1941; *Proceedings of the Society for Experimental Biology and Medicine*, June 1941.

[3] *The Journal of the American Medical Association*, March 1, 1941; *American Journal of Obstetrics and Gynecology*, July, 1941 and December, 1941.

and these have led to very important results, amongst them a completely new conception of *erythroblastosis fœtalis*. Both groups of workers have examined a very large number of men and women, and have found that the Rh factor is present in about 85% of the population and absent in about 15%. They have further shown that the Rh factor is capable of producing anti-Rh agglutinins in the human blood and have actually found these agglutinins in a considerable number of cases. Transfusion accidents of the first type mentioned above can now be explained as follows. A patient needing repeated transfusion and who is "Rh negative" is likely to be given blood containing the Rh factor, as 85% of the donors are "Rh positive". As normally anti-Rh agglutinins are not present in the blood, the first transfusions in such a case will pass without accident. In the course of time, however, anti-Rh agglutinins will be formed in the blood of such patients and in a later transfusion a more or less severe reaction will occur. That this explanation is correct has been shown by the successful use of "Rh negative" blood for patients who had previous transfusion accidents.

Transfusion accidents in pregnancy also will concern "Rh negative" mothers. On account of the general distribution of the Rh factors (85% positive, 15% negative) it is likely that the husband is "Rh positive". As it has been shown that the Rh property is inherited as a dominant factor, all or half the children of such a marriage will be "Rh positive", depending on whether the factor in the father is homozygous or heterozygous. If an "Rh negative" woman harbours in her uterus an "Rh positive" fœtus, it is possible that anti-Rh agglutinins are formed in her blood. If circumstances then arise which necessitate a transfusion, and she receives blood containing the Rh factor, a more or less severe reaction will occur even on the first occasion. The practical result of these deliberations is to exclude the husband from the list of possible donors whenever there is the slightest suspicion that the Rh factor may be of importance. What circumstances might lead to such a suspicion will be seen later.

The development of an "Rh positive" fœtus in an "Rh negative" mother may have further consequences. In the same way as it is possible for agglutinogens to pass from the fœtal to the maternal circulation it is possible for anti-Rh agglutinins formed in the maternal blood to leak back into the fœtal circulation. It seems that the discovery of this process has led to the solution of the problem of *erythroblastosis fœtalis*. This condition, which clinically occurs in three different forms, *hydrops fœtalis*, *icterus gravis* and hæmolytic anæmia of the newborn, is characterized in all its forms by the presence of numerous nucleated red cells in the fœtal circulation and widespread persistence of extramedullary blood formation. It was generally considered to be some kind of blood or bone marrow disease. The puzzling fact was that the condition completely disappeared when it was possible to tide the child over the first few weeks of life by repeated transfusions, a fact not quite consistent with a primary blood or bone marrow disease. A reasonable explanation now seems possible. The leakage of anti-Rh agglutinins from the maternal into the fœtal circulation leads to a continuous destruction of fœtal red blood cells. The extramedullary hæmatopoiesis is nothing but the response of the body to the excessive loss of red blood cells. The occurrence of immature red blood cells in the circulation discloses the incapability of the body to provide a sufficient number of mature red cells for the circulation. In the neo-natal life the agglutinins present at birth will continue their work till they are exhausted, and if the child is tided over this period by transfusions of blood which in all likelihood will not contain anti-Rh agglutinins, the destruction of red cells will cease and the condition become normal. The occurrence of some form of erythroblastosis in practically all the children of certain families would be well accounted for on the basis of these arguments. Levine and his collaborators have gone even further by including in the possible consequences of such iso-immunization certain cases of abortion, accidental hæmorrhage and so on.

What practical consequences can be drawn from these discoveries? Immune serum which would allow a determination of the Rh factor by the usual methods seems to be not available except in the laboratories of the two groups of workers. However, both groups[1,2] have reported on a special cross-matching test which should help to overcome the present difficulties. In the hands of certain Australian workers Levine's technique seems to give better results. It might be necessary to examine a large number of people with this technique when a donor is needed for a patient in whom transfusion accidents have already occurred, or for a pregnant woman who has been previously delivered of erythroblastotic babies. The extra work will be justified when suitable donors can thus be found for such a patient.

With regard to an erythroblastotic baby, it is self-evident that the mother should never be used as a donor. Such a procedure would introduce additional anti-Rh agglutinins into the baby's circulation. Strangely enough, however, better results seem to have been obtained when the baby is given blood from another "Rh negative" donor. The reason apparently is that "Rh positive" blood is likely to be destroyed by the anti-Rh agglutinins still present in the baby's circulation, while blood, "Rh negative" but free from anti-Rh agglutinins, would escape destruction.

Should a mother be allowed to breast feed her erythroblastotic baby? It is too early to be dogmatic about that, but it has been suggested that anti-Rh agglutinins, although not so widespread in body tissues and fluids as those of the ABO series, can pass into the milk. If this suggestion were substantiated, breast feeding would not be advisable.

It can be seen from this summary that some aspects of the problem raised by the discovery of the Rh factor need further elucidation or final confirmation; but a state has been reached at which the results of the well-established part of the work can and should be applied in the practical management of patients.

MYCOBACTERIUM TUBERCULOSIS.

POSSIBLY no bacterium has been given so much study as *Mycobacterium tuberculosis*. There is always something new to be learnt about this protean organism. R. A. Hunter has recently made a painstaking study of the appearances of *Mycobacterium tuberculosis* in sputum.[3] He states that there is "a general but vague belief that the tubercle bacillus is a branching form of micro-organism and therefore allied to the higher bacteria". But branching has rarely been observed, and this fact "engenders a feeling of doubt in the minds of many workers". Branching forms of the avian organism have been observed *in vitro*; but few if any similar examples of mammalian types have been noted. "It appears therefore that the assertion that tubercle bacilli belong to the streptotricheæ is based solely on branching forms obtained *in vitro* and probably of the avian type." Hunter carefully investigated the sputum of 50 patients who were voiding large numbers of tubercle bacilli. He found lateral buds or branches in 36 cases. In some the branching was bizarre and extensive. He states that to avoid disruption the sputum should be spread thickly and with no unnecessary vigour on the slide; drying and fixation should be done without undue heating; and methylated spirit should not be used for decolorizing. Picric acid solution should be used as a counter stain. Clumping of bacilli is believed by Hunter to occur because the organisms are in an unsuitable environment.

It appears that the long, filamentous, branching form of tubercle bacillus, is the consummate type and only obtainable under ideal parasitic conditions. Other forms are apparently adaptations engendered by the introduction of unsuitable or inhibitory factors.

Hunter's findings should prove of value in the study of tuberculosis.

[1] *Journal of Experimental Medicine*, October, 1941.
[2] *Proceedings of the Society for Experimental Biology and Medicine*, October, 1941.
[3] *Tubercle* November, 1941.

Abstracts from Medical Literature.

RADIOLOGY.

Dysostosis Multiplex.

M. J. HUBENNY AND P. J. DELANO (*American Journal of Roentgenology*, September, 1941) state that *dysostosis multiplex* is a congenital disease characterized by interference with ossification, which leads to degenerative changes in cartilage. The active stage of the dystrophy appears to cease as the time for fusion of the epiphyses and diaphyses approaches, but the pressure deformities remain as evidence that the dystrophy has existed. Prominent among the characteristic lesions are wedge-shaped deformities of the vertebræ, failure of appearance of epiphyseal centres and delay in their ossification, shortening of the neck due to cervico-dorsal deformity (which also leads to a shelf-like protrusion of the upper part of the thorax), and shortness of the trunk as compared with the limbs. Deformities of the skull are variable and inconstant; there may be scaphocephaly or oxycephaly and depression of the bridge of the nose. Corneal opacities are found in some cases. The differential diagnosis of the condition from polyarthritis of infancy, hypothyreoidism, Pott's disease, achondroplasia, Ollier's disease and Scheuermann's disease is discussed.

Funnel-Chest Deformity.

LESTER W. PAUL AND M. R. RICHTER (*American Journal of Roentgenology*, November, 1941) describe the clinical and anatomical aspects of funnel-chest deformity. Attention is called to a number of changes which allow its recognition in the frontal projection of the chest—a necessity when examination is confined to this view, as is common in mass surveys. Of particular importance is the fact that the loss of aeration commonly present in the medial portion of the base of the right lung may be confused with such pathological conditions as atelectasis, pleural thickening, chronic inflammatory disease or consolidation. A recognition of the mediastinal displacement phenomena and the changed contour of the anterior arcs of the ribs will help to prevent such misinterpretation.

"Blast Chest."

A. ROBINSON THOMAS (*British Journal of Radiology*, December, 1941) states that there is still some doubt as to the exact definition of "blast chest"; it may for the present be described as the effects on the thoracic cage and its contents of the high pressure waves following explosions. It is possible, however, that minor degrees of the same condition may be produced by direct violence, such as severe blows on the chest or crushing of the thoracic cage by falling masonry. The condition is caused by suction following the wave, by the raising of pressure in the bronchial tree by the wave, and by direct action of the wave on the thoracic wall. It seems probable that all these factors may play a part; but a combination of the effects of elevation of the pressure in the bronchial tree and of simultaneous compression of the thoracic wall (the compensating expansion that would in the ordinary way take place thus being prohibited) would account for the changes found experimentally, clinically and radiologically. It would appear that the pathological lesion in these cases is hæmorrhage to a greater or less degree into the lung alveoli, giving rise to areas of consolidation, and it seems that these areas cause the basic radiological appearance. This, as in other forms of consolidation of the lung, either from exudation or from transudation into the alveoli, is characterized by loss of radio-translucency, the size and density of the opaque area being determined by the extent of the lesion. It must, however, be remembered that the lesions mentioned are those sufficent to cause death, and that less extensive lesions are present in patients who recover; moreover, less extensive lesions may be complicated by fractured ribs, by hæmothorax, by hæmopneumothorax, by deflation of alveoli following inhalation of dust or other foreign bodies, and lastly, by true pneumonic consolidation following infection of damage to the alveoli by streptococci or pneumococci. All these complications will affect the radiological appearance of a "blast chest", and all must be taken into account in the report on such a case. The radiological picture may vary within wide limits, from a slight loss of translucency locally to a general opacity of the lung field. In addition to this there is, in the author's experience, a characteristic appearance of the ribs over the part affected, these ribs being crowded together posteriorly and widely separated anteriorly. That is to say, they are in a position of hyperexpansion of the chest as compared with the other ribs. Clinically, in mild cases of "blast chest" there are few or no physical signs, though inspection of the chest reveals hyperexpansion ("ballooning"), as would be expected from the radiological appearance of expansion. An X-ray examination is of importance, for without this lesions of some magnitude may be unsuspected, with unfortunate results later. On this account it is of the greatest importance that radiologists should be aware of the changes appearing in the chest, and should bear in mind that such changes may be small and easily missed unless care is taken.

Spontaneous Pneumoperitoneum: A Radiological Sign Found in the Supine Position.

LEO G. RIGLER (*Radiology*, November, 1941) states that in a series of cases of pneumoperitoneum he has observed a radiological sign which is particularly valuable, because it is manifested in the ordinary "scout" skiagrams of the abdomen made with the patient in the supine position. Essentially this finding consists in the ability to visualize on the film the outer as well as the inner wall of the bowel. It appears to occur only in those cases in which relatively large quantities of gas and some fluid have entered the peritoneal cavity. In such circumstances a sufficient quantity of contrast medium is present to separate the individual loops of bowel from each other. The loops themselves are usually greatly distended with gas because of the peritonitis that is present. As a result it is possible to observe the contour of both the inner wall of the bowel and of the outer wall, the thickness of the wall itself being quite apparent. In the ordinary case of ileus it is relatively easy to observe the inner wall of the bowel, because of the contrast between it and the gas which distends the bowel. Likewise, the thickness of the bowel wall can be approximately ascertained, because with two loops of bowel lying in close juxtaposition to each other, the gas filling each loop delineates its wall; thus the thickness between the two layers of gas ordinarily represents the thickness of both bowel walls. In case of effusion into the peritoneal cavity the diagnosis can often be made because of the increased thickness of the shadow separating the layers of gas, which results from the entrance of fluid between the two loops. In the case of pneumoperitoneum a different picture is presented. Gas enters in between the two loops, so that there is a definite separation by a medium of lower density, and the outer limits of the wall of each loop of bowel can thus be delineated. This is practically pathognomonic of pneumoperitoneum and is easily observed in ordinary films of the abdomen, the patient being in the supine position.

Patent Interauricular Septum Associated with Mitral Stenosis: Lutembacher's Syndrome.

GEORGE J. BAYLIN (*Radiology*, January, 1942) reports two cases of defective interauricular septum associated with mitral stenosis. Patency of the interauricular septum is relatively common, but the coexistence of stenosis of the mitral valve is rare. The latter condition, though it presents a varied clinical picture, produces a skiagram which is characteristic. The cardiac silhouette is enlarged to the right and left, with prominence of the pulmonary conus. The aortic shadow is usually normal or else actually smaller than normal. There is prominence of the hilar shadows, which are indeed so striking that they often resemble irregular lymph node masses. The left auricular border may be obscured by the bulging, dilated conus, but is often enlarged to the left. The picture, though characteristic, is nevertheless confusing. The densities in the hilar and conus regions have been diagnosed as intrathoracic tumours, and on one or two occasions patients have actually been subjected to exploratory operations. Fluoroscopy and oblique films will serve to clarify the changes observed in the conventional chest skiagram, and the anatomical relationships of the shadows can usually be readily established.

Patellar Anomalies: Radiological and Clinical Consideration.

HORACE C. JONES AND DONALD W. HEDRICK (*Radiology*, January, 1942) discuss the pathogenesis, diagnosis and treatment of patellar anomalies. They state that the differential diagnosis between patellar anomaly and fracture is important and is sometimes difficult. Usually it can be made accurately if the history and the physical and radiological findings are correlated. In cases of anomaly the trauma is usually indefinite and inadequate to explain the findings observed on the films. The clinical findings do not harmonize with the radiological observations. In cases of fracture the injury is adequate to produce the findings observed on the films, which are those associated with this condition. In anomaly the fragment is smooth and entirely bounded by cortical bone, but in fracture the outline of the fragment is serrated and only partially bounded by cortical bone. In anomaly the adjacent

surfaces of the fragments are uniformly separated throughout their entire length, but in fracture the space intervening between the fragments is of varying width. A fracture line is rarely seen in the upper and outer quadrant, and is usually unilateral, whereas in anomaly the fissure is almost always in the outer and upper quadrant and frequently is bilateral. Bilateral skiagrams of the knees are often helpful and may make it possible to establish the diagnosis beyond any reasonable doubt.

PHYSICAL THERAPY.

The Treatment of Lymphosarcoma with Radioactive Phosphorus.

J. M. KENNY, L. D. MARINELLI AND L. F. CRAVER (*American Journal of Roentgenology*, February, 1942) present a preliminary report on the treatment of lymphosarcoma with radioactive phosphorus. Lymphosarcoma almost always becomes a generalized disease involving lymph glands in widely separated areas. The ideal method of radiation treatment would be some form of systemic irradiation that would preferentially attack the scattered foci of disease. It has been demonstrated that in leuchæmia there is a selective absorption of orally administered radioactive phosphorus by tissue infiltrated with leuchæmia cells. This suggested that lymphosarcoma with its close link to leuchæmia and its usually high degree of radiosensitivity might be effectively treated by the oral administration of radioactive phosphorus. Small amounts, approximately 0·5 millicurie, of radioactive phosphorus in the form of a 1·5% solution of sodium phosphate were administered to a group of patients. Lymph nodes were excised from these patients from one to thirty-one days later, and after suitable preparation their radioactivity was measured and corrected for decay to the date of administration of the phosphorus. At the time the paper was written eighteen patients with lymphosarcoma had been or were being treated with radioactive phosphorus. Five patients had had complete regression of all evidence of disease and had been free of all recurrence for periods varying from one to eight months. One patient did not respond to the therapy, one died during treatment, and two showed partial regression. The remaining nine patients were still under treatment. These results seem to indicate that radioactive phosphorus is a useful and effective method of therapy in lymphosarcoma. Care should be taken to watch for damage to the bone marrow by frequent blood counts and perhaps marrow aspirations.

Radiation Therapy in Chronic Arthritis.

IN an editorial article (*Radiology*, November, 1941) reference is made to a recent report on the efficacy of X-ray therapy in rheumatic diseases by C. J. Smyth, R. H. Freyberg and W. S. Peck, working at the University of Michigan. In nearly every case there was comparable involvement in a pair of joints. It was possible, therefore, to treat one of the joints with irradiation and leave the corresponding joint untreated to serve as a control for a better evaluation of the therapeutic result. The only other therapy was of a general nature—rest, the administration of salicylates, the application of heat, exercise and treatment for anæmia. The X-ray technique consisted of the following factors: 200 kilovolts, filtered through 0·5 millimetre of copper and 1·0 millimetre of aluminium; 50 centimetres focal skin distance; the usual size of the field, approximately 15 centimetres square. Each field was treated three times, receiving 200 *r* at each sitting. The treatments were given on alternate days. Most patients received three series of treatments to the same part with an interval of four to six weeks between series. In order to study the psychic effect that might be associated with the irradiation, a lead screen was sometimes inserted to give protection from the rays without the patient's knowledge. The clinical results obtained in the various types of rheumatoid disease are recorded with regard to subjective and objective benefits. In rheumatoid arthritis it was found that 44% of the patients received no benefit whatever from the X-ray therapy. Objective evidence of benefit was present in 26% of cases, but was of significant grade in only 14%. In 21% of the cases the subjective improvement lasted less than one month. X-ray therapy appeared to confer no protection against subsequent acute exacerbations of the disease, as they occurred as frequently in the treated joints as in the untreated ones. X-ray examination of the joints demonstrated no anatomical improvement. In a later report from the same institution, Smyth, Freyberg, and Lampe discussed the results obtained with X-ray therapy in 52 cases of *spondylitis rhizomelica*. In 72% of the cases a significant degree of subjective benefit was obtained, and in 50% significant benefit was shown by objective clinical changes. The therapeutic results varied indirectly in proportion to the stage and extent of the pathological process. In most of the reported cases relief of pain has been the outstanding benefit. The course of the joint changes and ligamentous calcification has usually not been materially altered.

Irradiation of the Pituitary Gland in Posterior Lobe Hyperfunction Controlled by Biological Tests.

E. P. PENDERGRASS, P. J. HODES AND J. Q. GRIFFITH, JUNIOR (*American Journal of Roentgenology*, November, 1941) record their experiences in the treatment of hypertension and menstrual headache which was found to be associated with antidiuretic substance in the blood. They irradiated the pituitary glands of 41 patients, 31 of whom had high blood pressure. The remaining 10 patients complained of symptoms referable to the menopause or to menstruation. In most of the cases blood studies for antidiuretic substance gave positive results prior to irradiation. Repeated antidiuretic studies were then carried out for comparison at various periods during and after irradiation. The test used for antidiuretic substance in the blood was that described by Griffith, Corbit, Rutherford and Lindauer. X rays generated at 200 kilovolts and filtered through 0·75 millimetre of copper and 2·0 millimetres of aluminium were used in the treatment. These were applied to two temporal fields, from 50 to 200 *r* being given per day and alternately to the two fields. The total dose necessary seems to be from 400 to 500 *r*. Thirty-one patients with hypertension were treated; the irradiation was directed to the hypophysis. No attempt was made to select the patients for treatment. The only prerequisite was a positive reaction to antidiuretic tests. Ten patients with symptoms thought to be attributable to catamenial fluid retention were treated. In about two-thirds of the cases irradiation of the pituitary caused the antidiuretic substance to disappear from the blood. This suggests that there may exist in the region of the hypophyseal fossa a centre or centres which partially control blood antidiuretic substance. Of the 31 hypertensive subjects, eight showed considerable subjective improvement without any drop in blood pressure. In five cases the effect of irradiation was encouraging; the clinical symptoms disappeared and the blood pressure dropped considerably. Of five patients treated for intense menstrual headache, three experienced great relief of pain and the condition of one was moderately improved. As it has been noted by Cushing that patients who die of hypertension are found to have varying degrees of basophilic infiltration in the posterior lobe of the pituitary gland, it may be suggested that the beneficial effects noted in this group of patients were in part due to the effect of irradiation on the basophilic infiltration. As the group of patients examined is small, the authors regard the conclusions as being in the nature of a preliminary report.

Differentiation between the Direct and Indirect Effects of X Rays upon the Organs of Normal and Adrenalectomized Rats.

C. P. LEBLOND AND G. SEGAL (*American Journal of Roentgenology*, February, 1942) point out that it is now generally agreed that the primary effect of X rays on living tissues is one of destruction at the point of impact; but the possibility of secondary toxic effects must also be emphasized. They have attempted in an investigation to separate the specific and non-specific effects of X rays by examination of the organs located out of a narrow field of irradiation. Thus the thymus was examined when the abdomen was treated, the gastro-intestinal tract when the head was irradiated, and so on. In addition, because of the role of the adrenals in the response of the organism to toxic agents, all the experiments were repeated on adrenalectomized animals. Normal adult rats weighing approximately 150 grammes were used in the experiments. The results showed conclusively that large doses of X rays produced a secondary effect on organs distant from the area of irradiation—namely, constant thymo-lymphatic atrophy and adrenal hypertrophy and frequently fatty infiltration of the liver and ulcerations of the stomach. These lesions indicate the existence of a general intoxication following irradiation. It was also shown that after removal of the adrenals the thymo-lymphatic atrophy and fatty infiltration of the liver produced by an intense localized irradiation were suppressed, but the gastric lesions and general lethal effects of the rays were increased. Thymo-lymphatic atrophy is also produced by the direct impact of the rays. Lesions of the testicular epithelium are exclusively produced by such direct effect.

Special Abstract.

THE TREATMENT OF BURNS.

IN the *New Zealand Medical Journal* for April, 1942, there appears a paper by Dr. H. P. Pickerill on the subject of burns and their treatment. Dr. Pickerill begins by calling attention to opinions recently expressed, of which the following are the chief features. (i) Burns associated with total loss of skin should be regarded simply as large open wounds. (ii) Shock may be combated in other ways than by the use of tanning agents, and should be prevented rather than treated. (iii) Early skin grafting is desirable. He agrees that the tannic acid treatment, introduced by Drinkwater twenty years ago, must have saved thousands of lives, and that for burns of the second degree (and perhaps the first degree) it is still probably unrivalled; but he considers that when the full thickness of the skin appears to have been destroyed or so damaged that it will not survive, tanning has its limitations. The chief of these are the following. (i) It is impossible to keep the area from becoming infected during the long period before the crust separates. (ii) Secondary toxæmia or septicæmia (usually caused by a hæmolytic streptococcus) often occurs and prolongs the patient's stay in hospital. (iii) The large, foul, suppurating area that often results is extremely difficult or impossible to heal by grafting, and if it heals without grafting it leads to disfiguring cicatrices, which may also greatly limit function. Dr. Pickerill thinks that there is only one way of preventing a large open wound from becoming infected, and that is by covering it with a living sheet of the patient's own skin; this should be done as early as possible. The local use of sulphanilamide makes it possible now to graft unsterile areas in which grafts were previously apt to fail. J. M. Converse recommends that at the time of primary excision of all devitalized tissue the wound should have sulphanilamide powder blown into it in a thin, even layer. A skin graft is then laid into the wound and held in place by firm, even pressure from cotton-wool or sponge. (Dr. Pickerill admits that this method is not efficacious if the area is highly purulent.) The invention of the dermatome makes it possible to cut large sheets of skin from the abdomen, back or buttocks; the sheets are of predetermined thickness, and "take" with certainty. If the patient's burned area is so great that he cannot spare sufficient skin, other means have to be used. American surgeons have successfully used refrigerated skin from the backs of recently dead newborn babies. Staige Davis has proved that skin grafts can be kept in oil in a refrigerator for three weeks without losing their vitality. Dr. Pickerill refers to the general disinclination towards immediate excision of necrotic tissue in burns, and suggests that one reason is the fear of removing some islands of skin that might possibly survive and spread. When it is a question of saving the patient's life and preventing contractures, such islands of skin are of no importance; moreover, the dermatome does away with this objection. In the case of burns so infected at the first examination that immediate grafting is obviously unwise, the necrotic tissue is removed, a sulphanilamide closed pressure dressing is applied and the grafting is carried out at the earliest subsequent opportunity. Dressings should be done with full operating theatre technique to prevent infection.

Referring to infection, Dr. Pickerill points out that immediately after being burnt the tissues are more sterile than they are ever likely to be again. Efforts should be made to prevent the infection of burns by the application of unclean household remedies and rags. He advocates educational propaganda to urge that people who have sustained serious burns should be "rushed to hospital". Contamination may also be caused by the hands of nurses and house surgeons in the casualty department, and from droplet infection. A third source of infection may be the skin surrounding the burnt area. Advances in surgery during the past twenty years that are of use in the prevention of infection of burns are the following: (i) the local use of sulphonamide drugs; (ii) the "closed" treatment of wounds; (iii) the excision of wounds rather than *débridement*; (iv) prolonged local anæsthesia with extremely dilute solutions; (v) local refrigeration; (vi) the dermatome.

Turning to the treatment of shock, Dr. Pickerill suggests that tannic acid "held the field" for so long because it prevented the patient from dying of shock, and he points out that some other means of achieving this object is necessary before the tannic acid method is replaced. He classifies shock associated with burns into two types, each different in origin; the first type is brought about by the arrival at the sensory nervous system of "an overpowering number of acute sensory stimuli" from the burned nerve endings; the second type (occurring from twelve to forty-eight hours later) is due to absorption of large quantities of toxic material (histamine, a histamine-like substance or some by-product of acute necrosis) from the damaged tissues. The second type of shock will kill the patient unless this is prevented. The first type of shock is best treated by Crile's method of local anæsthesia. Morphine may be used; but this drug merely dulls the perception of the stimuli, and a considerable degree of primary shock may persist. Dr. Pickerill quotes work in Russia on this subject, which has been successful.

> In Russia great success is claimed with the use of two solutions. These are procain 1 in 400 combined with percain 1 in 4,000, and also sovkain (percain) 1 in 10,000, used up to 1,000 c.c., all the surrounding tissue being infiltrated. Anæsthesia lasting from 5 to 6 hours is obtained.
>
> Vishnevskey further claims that "a massive infiltration of the tissues with procain prevents the rapid absorption of toxic products of decomposed tissue", and preliminary local anæsthesia is regarded as part of the preoperative treatment of shock.

Another method of combating shock is the use of local refrigeration—the local cooling of necrotic tissue. It is essential that the temperature should be kept well above freezing point; F. M. Allen, who originated the method, advocates a temperature slightly above or below 5° C. Allen summarized the advantages of refrigeration as follows:

> . . . by refrigeration one may obtain complete anæsthesia of all tissues in a few hours. Instead of providing only anæsthesia of the nerves, refrigeration inhibits all protoplasmic activity including the production of shock. In this manner one is permitted to operate in a bloodless field, i.e., the refrigerated tissues will remain in that state long enough to permit the performance of any ordinary operative procedure. The wound does not decompose or become infected because the low temperature also inhibits the action of enzymes and bacteria.

By way of summarizing the opinions of the various workers whom he quotes, Dr. Pickerill states that ". . . in effect, refrigeration fights a strong delaying action, permitting a proper surgical frontal attack on the area". Refrigeration in burns has three purposes: (i) to prevent primary shock if it can be applied soon enough, (ii) to prevent the absorption of shock-producing substances, (iii) as an accessory anæsthetic agent for operative procedures. If the patient comes under medical care early enough, it seems that both stages of shock may be totally prevented by the use of dilute local anæsthetic agents and by refrigeration. Established shock must be treated by the usual methods (the administration of morphine, oxygen, adrenal cortex and histaminase, the application of heat, and blood transfusion). Dr. Pickerill lays stress on his belief that the idea that burns and shock are inseparable is the greatest obstacle to progress in the treatment of serious burns. Tanning is much slower than refrigeration in retarding the absorption of shock-producing substances. If tanning is discouraged on the face or hands or in the flexures, Dr. Pickerill wonders why it should be used in any other area. There is evidence to show that liver damage follows absorption of toxins from burnt tissue, and general anæsthesia may add to this damage; therefore he considers general anæsthesia contraindicated. Oxygen may be of value in the prevention of shock. The excision of necrotic tissue, he holds, is only the application of general surgical principles, and again he urges that a burn should be treated as an open wound, of which the superficial layers have been severely traumatized. The tissue is a mass that is forming highly dangerous toxins threatening the patient's life, and sound surgical principles dictate its complete and immediate excision. Dr. Pickerill refers to Kisima's work on histamine in burns and on changes in the adrenals. He quotes the following statements:

> There is a decrease in the chromaffin and in the amount of adrenalin in the adrenal medulla in both burns and histamine poisoning. The clinical signs and histological changes are similar in both conditions. Histamine is probably an important factor in the toxin production in burns, and alterations in the adrenal glands are the probable cause of death in the fatal cases.

These he considers to provide further arguments in favour of primary excision and primary skin grafting in severe burns.

Naval, Military and Air Force.

APPOINTMENTS.

THE undermentioned appointments, changes *et cetera* have been promulgated in the *Commonwealth of Australia Gazette*, Number 146, of May 21, 1942.

Citizen Naval Forces of the Commonwealth.
Royal Australian Naval Reserve.

Appointment.—John Russell Robert Taylor is appointed Surgeon Lieutenant, dated 25th March, 1942.

AUSTRALIAN MILITARY FORCES.

Australian Army Medical Corps.

The provisional appointments of Captains N274198 G. N. M. Aitkens, N274914 M. N. Tinckam, N274199 A. E. H. Salter, N27248 J. G. Buckley, N274181 D. C. Williams, N278473 A. J. Shappere, N27148 W. J. Pullen and N278469 C. W. Coombe are confirmed.

Captain N273245 H. G. B. Bruce is transferred, 29th January, 1942.

Captain (provisionally) E. F. Fletcher is transferred to the Reserve of Officers, 13th March, 1942.

The date of appointment of Lieutenant (Temporary Major) H. S. Jacobs, V.D., which appeared in Executive Minute No. 51/1942, promulgated in *Commonwealth Gazette*, No. 79 of 1942, is amended to read "12th March, 1942".

The following officers are appointed from the Reserve of Officers: Captain V9321 N. A. Maclure, 3rd December, 1940, and Honorary Captains V10876 A. L. B. Webb and S. Finklestein and to be Captains (provisionally), 18th February, 1941, and 17th April, 1942, respectively.

To be Majors (temporarily).—Captains N283610 A. P. Gunning, 14th April, 1942, and W32451 J. A. Love, 16th April, 1942.

To be Captains (provisionally).—Hartley Willison Austin, Stuart Fraser and William Harold James Cole, 22nd April, 1942; Maurice Beraha, James Buchanan Alexander, Joseph James Hurley, Graham George Godfrey, Francis James Bryan, Lindsay Charles Brittingham, Richard Maxwell Biggins and Phillip Gerrard Dowling, 24th April, 1942; and Maurice Curzon, 28th April, 1942.

Honorary Captain H. S. Moroney and Captain B. T. Mayes are retired, 27th February, 1942, and 23rd March, 1942, respectively.

To be Honorary Captains.—Andrew John Hakendorf, 13th April, 1942; Keith Ewan Harrison, 15th April, 1942; Mervyn Everard Hay Elliott and Ian Monk, 20th April, 1942.

Major N393096 A. McKillop, D.S.O., is appointed from the Reserve of Officers, 16th March, 1942.

The provisional appointment of Captain N57446 A. A. Callen is confirmed.

Lieutenant (on probation) (Temporary Captain) V81364 J. H. Gale ceases to be seconded, is transferred and retains the temporary rank of Captain, 9th March, 1942.

Captains (provisionally) G. H. Ellis and J. A. M. Shera are transferred, 8th April, 1942.

The following officers are transferred from the Australian Imperial Force: Captains NX76540 S. D. Allen, NX76501 N. J. Clements, NX76539 F. M. C. Jones, NX76470 J. M. McDonald, NX76543 F. O. B. Wilkinson, NX77177 P. Berger and NX76363 H. B. Gatenby, 1st March, 1942; and VX65513 R. L. Sleeman, 6th March, 1942.

The following officers are transferred to the Reserve of Officers: Major W3610 W. P. White and Captains (provisionally) W34897 N. C. Joel, A. B. Webster, P. C. Hogan and B. O. Bladen, 31st March, 1942.

Major N383089 S. U. Gentile is placed on the Retired List, 7th April, 1942.

Major J. S. Reid is appointed from the Retired List, 25th March, 1942.

The following officers are appointed from the Reserve of Officers and to be Captains (provisionally): Honorary Captains A. S. Saleeba, 15th January, 1942; N278541 G. A. Chambers and R. MacQueen, 5th February, 1942; N101319 C. H. Wood, 25th March, 1942; J. M. O'Donnell, 4th April, 1942; R. F. Back, A. S. B. Studdy and M. A. Radcliffe-Taylor, 9th March, 1942.

To be Major (temporarily).—Captain (provisionally) Q123569 B. J. Butcher, 15th April, 1942.

To be Captains (provisionally).—Frank Spence Sedgwick and Lesley Vincent, 13th April, 1942; Francis Patrick Guilfoyle, 15th April, 1942; William Blair Christie Gray, 16th April, 1942; Joseph Roy Baker, 17th April, 1942; Thomas Arthur Glennie Holmes, John Chatteris Bell Allen, John Stephen MacMahon, Francis Vincent Twohig, Malcolm Denholme Hunter Harpur, Reuben Segal, Colin Robert Moore Laverty, Beaumaris William Stevenson, John Joseph Donnellan, Horace Herbert O'Connor, Percie Leonard Charlton, Richard St. John Honner, John William Quilter, 18th April, 1942; Erwin Jan Haberfield, 20th April, 1942, and Leopold Winthrope, 21st April, 1942.

Honorary Captain N282309 H. F. Alsop is placed on the Retired List, 2nd April, 1942.

The name Marion Aroha Redcliffe-Taylor, which appeared in Executive Minute No. 227/1940, promulgated in *Commonwealth Gazette* No. 248 of 1940, is amended to read "Marion Aroha Radcliffe-Taylor".

To be Honorary Captains.—George Hedley Kennett, Noel Francis George and John Frederick Briton Langdon, 9th April, 1942, John Love Steele Scott and Margaret Stewart, 13th April, 1942, and Preston Guy Reynolds, 15th April, 1942.

ROYAL AUSTRALIAN AIR FORCE.

Citizen Air Force: Medical Branch.

Bruce Toomba Mayes, M.B., B.S., F.R.C.S., F.R.A.C.S. (4848), is appointed to a commission on probation with the rank of Flight Lieutenant with effect from the 23rd March, 1942.

Reserve: Medical Branch.

The following are appointed to commissions on probation with the rank of Flight Lieutenant with effect from 27th March, 1942: Reginald Ernest Braithwaite Brown, M.B., Ch.M. (4854), Leslie Elmo Goldsmith, M.B., B.S. (4853), Howard Hoban, M.B., B.S. (4852), Francis James Kenny, M.B., B.S. (4851), Nicholas Morrissy, M.B., B.S. (4850).—(Ex. Min. No. 66—Approved 20th May, 1942.)

CASUALTIES.

ACCORDING to the casualty list received on May 27, 1942, Captain R. A. Spence, A.A.M.C., Cremorne, New South Wales, is reported to have died of injuries.

According to the casualty list received on May 28, 1942, Lieutenant-Colonel E. L. Cooper, A.A.M.C., South Yarra, Victoria, is reported to have died of illness.

Correspondence.

CRUSH INJURY AND RENAL FAILURE.

SIR: With reference to my article on "Collapse and Renal Failure", which appeared in your issue of November 15, 1941, page 569, in which I endeavoured to prove that the renal damage in cases of so-called "crush injury" is in all probability due to anoxia. I shall be obliged if I may here enumerate some considerations which weigh against the acceptance of the view that the renal damage in "crush injury" is due to some obscure toxic derivative of crushed or necrotic muscle or tissue, and which were omitted from my original article.

1. The renal damage in cases of "crush injury" appears early, and is progressive even though the crushed limb be amputated.[1]

2. It is not possible to establish by cross-circulation or other methods that the blood of an animal suffering from traumatic shock is toxic.[2]

3. The characteristic degeneration of the epithelium of the renal tubules in "crush injury" is not found in infarction or absorption of blood clot (tissue necrosis).

4. The renal damage met with in "crush injury" is found in cases of injury where no crushing of the skeletal muscles has occurred.[1][2][3]

Yours, etc.,

J. WALKER TOMB.

Bangalla,
Cherry Street,
Warrawee,
New South Wales.
May 19, 1942.

References.

[1] E. G. L. Bywaters and D. Beall: "Crush Injuries with Impairment of Renal Function", *British Medical Journal*, March 22, 1941, page 432.
[2] R. J. S. McDowall: "The Circulation in Relation to Shock", *British Medical Journal*, June 8, 1940, page 919.

(42) A. M. Glen: "Temporary Vascular Occlusion ending Fatally in Uræmia", *British Medical Journal*, December 20, 1941, page 875.
(43) J. Young and J. McMichael: "Crush Syndrome in Obstetrics", *British Medical Journal*, December 20, 1941, page 887.
(44) R. Mayor-White and O. M. Solandt: "A Case of Limb Compression Ending Fatally in Uræmia", *British Medical Journal*, Volume I, 1941, page 434.

Notice.

THE following letter has been received by the Medical Secretary of the Victorian Branch of the British Medical Association, who has asked for its publication in this journal.

LIQUID FUEL CONTROL BOARD, VICTORIA.

Exhibition Buildings,
Rathdown Street,
Carlton, N.3.

Dr. C. H. Dickson,
Medical Secretary,
British Medical Association,
Albert Street,
East Melbourne, C.2.

Dear Sir,

As you would probably be aware, this Board is faced, from time to time, with applications for additional quantities of petrol to enable persons to travel to country centres for health reasons. Some of these applications involve very considerable quantities of petrol.

Many of the applications, if not actually frivolous, are, at best, based upon considerations of personal convenience only, and, although in all cases we require that medical certification will be submitted, the nature of this certification when received is not always conclusive.

It is often said, for instance, that the applicant is, or has been, suffering from such-and-such a complaint, and that a temporary change of residence would be beneficial. A statement of this nature is not very helpful, and it is often found necessary to refer the matter back to the practitioner for further advice.

It is suggested that your Association might be prepared to assist the Board by communicating with doctors advising them that, in matters of this kind, it is not sufficient merely to give certification as to the applicant's past or present illness, but that responsibility should be accepted for stating clearly whether or not the person concerned is quite unfit to travel by rail or other public transport, or whether such travel, even if undertaken, would be definitely prejudicial to the patient's present or future condition.

If your Association is prepared to act in this way, the Board would, of course, be extremely grateful.

Yours faithfully,
E. V. FIELD,
Secretary.

Obituary.

ANDREW BUCHANAN STEELE.

WE regret to announce the death of Dr. Andrew Buchanan Steele, which occurred on May 22, 1942, at Brisbane, Queensland.

Nominations and Elections.

THE undermentioned have applied for election as members of the South Australian Branch of the British Medical Association:

Heddle, Robert Charles, M.B., B.S., 1942 (Univ. Adelaide), 32, Thornber Street, Unley Park.
Verco, Peter Willis, M.B., B.S., 1942 (Univ. Adelaide), 7, Fitzroy Terrace, Prospect.

The undermentioned has been elected a member of the South Australian Branch of the British Medical Association:

Cherry, Alan Percival, M.B., B.S., 1941 (Univ. Adelaide), 23, Brougham Place, Alberton.

The undermentioned have applied for election as members of the New South Wales Branch of the British Medical Association:

Cumberland, Victor Henry, M.B., B.S., 1941 (Univ. Sydney), King George V Hospital, Camperdown.
Macintosh, Neil William George, M.B., B.S., 1933 (Univ. Sydney), Imperial Service Club, Barrack Street, Sydney.

Diary for the Month.

JUNE 9.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
JUNE 9.—Tasmanian Branch, B.M.A.: Branch.
JUNE 12.—Queensland Branch, B.M.A.: Council.
JUNE 16.—New South Wales Branch, B.M.A.: Ethics Committee.
JUNE 17.—Western Australian Branch, B.M.A.: Branch.
JUNE 18.—New South Wales Branch, B.M.A.: Clinical Meeting.
JUNE 23.—New South Wales Branch, B.M.A.: Medical Politics Committee.
JUNE 25.—New South Wales Branch, B.M.A.: Branch.
JUNE 25.—South Australian Branch, B.M.A.: Branch.
JUNE 26.—Queensland Branch, B.M.A.: Council.
JUNE 26.—Tasmanian Branch, B.M.A.: Council.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment mentioned below without having first communicated with the Honorary Secretary of the Branch concerned, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

New South Wales Branch (Honorary Secretary, 135, Macquarie Street, Sydney): Australian Natives' Association; Ashfield and District United Friendly Societies' Dispensary; Balmain United Friendly Societies' Dispensary; Leichhardt and Petersham United Friendly Societies' Dispensary; Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney; North Sydney Friendly Societies' Dispensary Limited; People's Prudential Assurance Company Limited; Phœnix Mutual Provident Society.

Victorian Branch (Honorary Secretary, Medical Society Hall, East Melbourne): Associated Medical Services Limited; all Institutes or Medical Dispensaries; Australian Prudential Association, Proprietary, Limited; Federated Mutual Medical Benefit Society; Mutual National Provident Club; National Provident Association; Hospital or other appointments outside Victoria.

Queensland Branch (Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17): Brisbane Associated Friendly Societies' Medical Institute; Bundaberg Medical Institute. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY HOSPITAL or position outside Australia are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.

South Australian Branch (Honorary Secretary, 178, North Terrace, Adelaide): All Lodge appointments in South Australia; all Contract Practice appointments in South Australia.

Western Australian Branch (Honorary Secretary, 205, Saint George's Terrace, Perth): Wiluna Hospital; all Contract Practice appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

All communications should be addressed to the Editor, THE MEDICAL JOURNAL OF AUSTRALIA, The Printing House, Seamer Street, Glebe, New South Wales. (Telephones: MW 2651-2.)

Members and subscribers are requested to notify the Manager, THE MEDICAL JOURNAL OF AUSTRALIA, Seamer Street, Glebe, New South Wales, without delay, of any irregularity in the delivery of this journal. The management cannot accept any responsibility unless such a notification is received within one month.

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